A STUDY OF UNILATERAL PROPTOSIS

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CERTIFICATE

This is to certify that the dissertation entitled **" A STUDY OF UNILATERAL PROPTOSIS"** presented herewith by ***Dr. P. SANTHI*** to the faculty of Ophthalmology, The Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the requirement for the award of M.S. degree in Ophthalmology is a bonafide work carried out by her under my direct supervision and guidance.

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DECLARATION

I, Dr.P. SANTHI, solemnly declare that the dissertation titled "A STUDY OF UNILATERAL PROPTOSIS" has been prepared by me.

This is submitted to The Tamilnadu Dr.M.G.R.Medical University, Chennai, in partial fulfillment of the regulations for the award of MS degree Branch [Ophthalmology].

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PART – I

INTRODUCTION

The orbit houses the eyeball and subserves the major functions of protection of the eye and facilitates extreme degree of motility possible by the eye. The orbit is pear shaped cavity, the stalk of which is the optic canal.

The orbital diseases gain importance due to involvement of optic nerve hence causing decreased vision. The intraorbital portion of optic nerve is longer (25mm) than the distance between, the back of the globe and optic canal (18mm). This allows for significant forward displacement of the globe (i.e) proptosis.

Clinically given case of unilateral proptosis is challenging even for experienced ophthalmologist with special interest in orbital diseases. Since orbit is in close relation to paranasal sinus, maxilla and cranium, primary diseases of these areas may initially present as proptosis. Hence a detailed study of, age of occurance of disease, findings on examination, progression of disease, available incidence of such disease, investigation results, become essential to diagnose a case of proptosis.

Proptosis is the hall mark of orbital diseases. Prompt and early diagnosis becomes essential as this socket, a small space in between orbital wall will lead to increased damage than one expects, and removal of masses will become difficult in later stages.

In unilateral proptosis cosmetic disfigurement becomes alarming to patients. Hence a detailed study of unilateral proptosis is necessary to find the diagnosis as early as possible so as to save the globe for cosmetic appearance and more than that to restore vision to its maximum possible limits.

This study aims to evaluate in detail variations in incidence, age group involved, etiology, methods of investigation and treatment offered to patients, and the results obtained.

ANATOMY

It is important to understand the relations of orbit and its neighbouring structures. These include the nasal cavities, accessory sinuses, communication with the interior of the cranial cavity by optic foramen and sphenoid fissure.

The orbital cavities are a pair of large bony cavities containing the eyeball, associated muscles, nerves, vessels, fat and lacrimal apparatus, the cavity is pear shaped and its apex directed posteriorly, medially and slightly upward.

The medial wall runs anteroposteriorly parallel to sagittal plane, lateral wall diverges at an angle of 45°, seven individual bones form the orbit namely maxilla, palatine, zygomatic, sphenoid, frontal, ethmoid and lacrimal.

ORBITAL MARGINS

The orbital margin is quadrilateral in shape with rounded corners. In adult orbital margin is wider than it is high.

SUPRA ORBITAL MARGIN:

Formed by frontal bone having sharp lateral two thirds and a rounded medial third. At the junction of the two areas is supra orbital notch or foramen for passage of supra orbital vessels and nerves.

INFRA ORBITAL MARGIN:

Formed laterally by zygomatic bone medially by maxilla.

LATERAL MARGIN:

Strongest part of orbital margin formed by frontal process of zygomatic bone below and zygomatic process of frontal bone above.

MEDIAL MARGIN:

Formed above by maxillary process of frontal bone, below by lacrimal crest of frontal process of maxilla.

The orbital width is greater than height usually. The relation between the two is given by orbital index.

$$OI = \frac{\text{Height}}{\text{Width}} \times 100$$

1. Megaseme : More than 89 →yellow races, orbital opening in round.
2. Mesoseme : 83-89 →white race (European)
3. Microseme : Less than 83→Black races orbital opening is rectangular[49].

WALLS OF ORBITAL CAVITY

The walls of orbital cavity are limited with periosteum and consists of a roof, a floor and a medial and lateral wall.

ROOF:

Is concave and separates orbital cavity from anterior cranial fossa and the frontal lobe of the cerebral hemisphere. Anteromedially roof is invaded by frontal air sinus and antero laterally by orbital part of lacrimal gland.

FLOOR:

This inferior wall is formed by orbital plate of maxilla and orbital surface of zygomatic, and orbital process of palatine bone separates orbital cavity from maxillary sinus. Floor is continous with lateral wall anteriorly but separated from it posteriorly by inferior orbital fissure. Inferior orbital groove is continuous with it and transmits so named artery and nerve[48].

LATERAL WALL:

The thickest wall anterior third is formed by the zygomatic bone, which separates the orbit from the temporal fossa, posterior two third formed by greater wing of sphenoid. Lateral wall and roof are continous anteriorly, but separated posteriorly by superior orbital fissure, this fissure communicate with middle cranial fossa. Marginal tubercle lies on frontal process of zygoma and gives attachments to levator palpebrae superioris, lateral palpebral ligament, lateral check ligament.

MEDIAL WALL:

This wall is formed by four bones. Frontal process of maxilla, lacrimal bone, orbital plate of ethmoid and body of sphenoid. Seperated from ethmoidal sinus. Anterior part of medial wall has the groove for lacrimal sac, formed by frontal process of maxilla anteriorly, and lacrimal bone posteriorly. Groove is bounded in front and behind by anterior and posterior lacrimal crest.

OPENINGS INTO ORBITAL CAVITY AND THE STRUCTURES THAT PASS THOUGH THEM

1. The main orbital: opening bounded by orbital margin

2. Optic canal: Lies in the lesser wing of sphenoid, measures 4-10mm long, connects middle cranial fossa with orbital cavity, transmits optic nerve with sleeve of meanings, ophthalmic artery with sympathetic plexus.

3. Superior orbital fissure: Lies between lesser and greater wings of sphenoid, connects middle cranial fossa with orbital cavity, lies between roof and lateral wall. Widest part is the medial end, medial end bridged by common tendinous ring. Transmits cranial nerves III, IV and VI; first (Ophthalmic division of cranial nerve V and sympathetic nerve fibres. Most of the venous drainage from the orbit passes through this fissure by way of superior ophthalmic vein to enter cavernous sinus.

4.Ethmoidal foramina: Lies in fronto ethmoidal suture

5.Anterior ethmoidal foramen: transmits anterior ethmoidal nerve and artery, opens into anterior cranial fossa.

6.Posterior ethmoidial foramen: traverses ethmoid bore, transmits posterior ethmoidal nerve and artery.

7. zygomatic facial and zygomatictemporal foramina : in lateral wall, transmits nerves and artery named similarly.

8.Inferior orbital fissure: Lies between lateral wall and floor of orbit communicates with pterygopalatine and inferotemporal fossa transmits infra orbital nerve, zygomatic nerve, communication between inferior ophthalmic vein and ptergyoid plexus. Lies between maxilla and palatine bone anteriorly and greater wing of sphenoid posteriorly.

SPACES IN THE ORBIT

From the surgical point of view there are four spaces which are relatively self contained, with each of which inflammatory process are contained for a considerable time and each of it must be opened separately.

1. Subperiosteal space: Between bones of orbital wall and periorbita.

2.Peripheral orbital space: Between periorbita and extra ocular muscles, which are joined by fascial connections making a more or less continuous circular septum, lesions produce eccentric proptosis[88].

3. Central space: A cone shaped area enclosed by the muscles (muscle cone), lesions produce axial proptosis.

4.Tenons space: around the globe between eyeball and tendons capsule.

RELATIONS OF THE BONY ORBIT

Superior relation:

Roof separates from meanings and frontal lobe of cerebral hemisphere.

Inferior relation:

Maxillary air sinus, the infra orbital canal.

Lateral relation:

Separates from temporal fossa containing temporalis muscle and prosteriorly from middle cranial fossa meninges and temporal lobe of cerebral hemisphere.

Medial relation:

Separates orbital cavity from nasal cavity, ethmoidal sinuses and sphenoidal sinus.

Dimensions:

Height of opening	35mm
Width of opening	40mm
Depth of orbit	40mm
Volume of orbit	30cc

Embryology of orbit:

Develops from the condensation of the mesoderm surrounding the optic vesicle and the stalk. Roof from the mesodermal capsule of the forebrain. Major part of floor and a small part of lateral wall formed by maxillary process.

DEFINITION, RELATED TERMS AND ITS DIFFERENTIATION

PROPTOSIS:

Means propulsion of eye ball and ptosis of lid, the propulsion of eye ball is passive, and usually occurs unilaterally.

EXOPHTHALMOS:

Means active protrusion of eye ball and retraction of the lids, and it is usually applied for proptosis, associate with dysthyroid state.

PSUEDO PROPTOSIS:[46]

There is no real forward displacement of the globe, certain orbit and ocular asymmetries may lead to false impression of protosis occurs in

1. Enlarged globe
2. Asymmetry of bony orbit
3. Asymmetry of lid fissure
4. Relaxation of recti muscle
5. Retracted lids
6. Enophthalmos of other eye

LUXATION OF GLOBE:

Ultimate stage of proptosis where the eyeball protrudes so far out of the orbit that the lids close behind it.

TYPES, AETIOLOGY AND CLASSIFICATION

1. Proptosis in divided into bilateral, unilateral, acute, intermittent, pulsating.

2. It may be axial, eccentric.

3. Unilateral proptosis may be

- Rapidly progressive - Orbital cellulities, Rhabdomyosarcoma, Hemorrage.
- Slowly progressive -Benign neoplasms
- Periodic exacerbation to remission- Lymphangioma
- Intermittent proptosis - Orbital varices, lymphangioma, Recurrent orbital hemorrage, Recurrent orbital emphysema
- Pulsuting proptosis - High vascular orbital tumor, AV malformations

AXIAL PROPTOSIS

AV Malforamations, Optic nerve glioma/meningioma/neurilemmoma, Metastatic tumors

ECCENTRIC PROPTOSIS

Downward: Neurofibroma, Lymphoma, Thyroid ophthalmology, Frontal mucocele, Neuroblastoma, Schwannoma

Down and in: Lacrimal gland tumor, Dermoid orbit, Psuedtumor orbit

Up ward: Maxillary sinus tumor, Lymphoma, Lacrimal sac tumor, Metastatic tumor

Lateral: Ethmoidal mucocele, Nasopharyngeal tumor, Midline Granuloma, Lacrimal sac tumor, Metastatic tumor

Medial: Lacrimal fossa tumor, Sphenoid wing meningioma

CLASSIFIED ACCORDING TO ETIOLOGY AS

1. **Congenital conditions:** Demoid cyst, congenital cystic eyeball, orbital teratoma
2. **Traumatic conditions:** Orbital hemorrage, retained intra orbital foreign body, traumatic emphysema
3. **Inflammatory conditions:** A**cute:** Orbital cellulites, abscess, panophthalmitis, cavernous sinus thromboses-later becomes bilateral, thrombophlebitis.

   Chronic: Psuedotumor, tuberculoma, gumma, sarcoidosis.
4. **Circulatory disturbances vascular lesion:** Angioneurotic edema, orbital varix, orbital aneurysms.
5. **Cyst of orbit:** Implantation cyst, parasitic cyst, haematic cyst.
6. **Tumors of orbit are primary, secondary, metastatic, mucoceles of paranasal sinuses.**

DESCRIPTION OF CAUSES

CONGENITAL AND DEVELOPMENTAL ANOMALIES

CRANIO FACIAL DYSOSTOSIS

Proptosis in this condition is the result of shallow orbit due to anterior displacement of greater wing of sphenoid[67]. In crouzon's and Apet's diseases ocular complications are related to coronal stenosis. Papilledema, optic atrophy and Strabismus are the other features.Other syndromes causing proptosis are Gruber's syndrome, Turner's syndrome (XO), Woife's syndrome (No.4 chromosome deletion), Morquio's disease (Hereditary osteochondro dystrophy).

CYSTIC TUMORS

Cystic tumors of the orbit are in many instances congenital. Although some of them do not become manifest until later years.Certain inflammatory and parasitic cysts may form in the orbit. Certain vascular neoplasms contain loculated cystic cavities[28].

TERATOMA

Contains multiple tissues which are representatives of more than one germinal layer. In addition to structures derived from ectoderm, tissues derived from endoderm and mesenchyme may be present.The cystic nature of these lesions is usually due to the presence of epidermoid cysts and embryonic

formations of mucin secreting gastro intestinal mucosa.Other cysts may be limited by ependymal cells or even choroid plexus.

ENCEPHALOCELE AND MENINGOCELE

Due to congenital dehiscences in the bones cerebral tissue herniates into the orbital cavity.If the meninges herniate causing a cystic tumor with cerebrospinal fluid, it is termed a meningocele.

If the brain protrudes inside the meningeal sac, then it is termed Encephalocele.

Attempts to explore the orbit may lead to rupture of the cyst causing cerebral damage or meningitis. Facial anomalies characterized by hypertelorism, broad nasal root and increased bitemporal diameter should alert the clinician to the possibility of an encephalocele.

MUCOCELE

Cystic structures lined with respiratory mucosa which form within the sinuses and often invade the orbit by eroding its bony walls. The cyst is filled with a thick mucoid secretion which is yellowish brown in color and jelly like in consistency. If the cyst is infected, it forms a pyocele.The mucoid lining of the cyst is of the respiratory type-pseudostratified ciliated columnar epithelium with goblet cells.They occur at all ages,but most cases are reported in patients over 45 years of age. The majority of orbital mucoceles arise in the frontal or ethmoidal sinuses and gradually erode their way into the orbit[85].

INFLAMMATION

ORBITAL CELLULITIS

Secondary to sinusitis, this is one of the most common causes of proptosis in childhood. The inflammatory process may spread from the adjacent sinus cavities into the orbit by means of communicating vessels or by direct erosion[34]. Toxic products of inflammation readily diffuse across the normally thin bony barrier that separates the sinuses from the orbit causing exudation from the orbital vessels. Inflammatory exudates may get loculated to form orbital abscess. In rare instances, it can cause cavernous sinus thromosis. Four cardinal signs are proptosis, lid swelling, chemosis and impaired ocular motility. This clinical picture is most often caused by ethmoiditis.

In recent years H.influenza has become the commonest cause of orbital cellulites in children[54].

TABLE I

DIFFERANTIATING POINTS BETWEEN EACH

Clinical features	Cavemous sinus thrombosis	Orbital cellulitis	Panophthalmitis
Laterality	unilateral initially then bilateral	Unilateral	Unilateral
Degree of proptosis	Moderate	Marked	Moderate
Vision	Not affected in early stages	Not affected in early stages	Complete loss
Cornea	Clear earlier	Clear earlier	Hazy
Ocular movements	Complete limitation	Marked limitation	Painful & limited
Edema in mastoid region	+	-	-
General symptoms with fever	Marked	Mild	Mild

PSEUDOTUMOR

Group of inflammatory disorders that produce a pseudo neoplastic orbital mass.

This causes pain, proptosis, chemosis and diplopia with visual loss to a virtually asymptomatic ill defined orbital swelling. Non specificity of histology findings, worsening of disease following biopsy and marked improvement with systemic steroids forms the hallmark of this condition. Pseudotumor can mimic thyroid exophthalmos. The clinical differentiating points are:-

TABLE 2

DIFFERENTIAL DIAGNOSIS OF PSEUDOTUMOR AND THYROID EXOPHTHALMOS[35].

NO	PSEUDOTUMOR	THYROID EXOPHTHALMOS
1.	No sex predilection	More common in women
2.	Often unilateral	Often initially or ultimately bilateral
3.	No systemic involvement	Systemic involvement
4.	Rapid progression	Less rapid progression
5.	Ptosis	Lid retraction
6.	Remission & exacerbation	Chronic course
7.	Steroid – low dosage	High dosage of steroids needed.

Specific disease entities can also cause pseudotumor in the orbit. There are Wegener's granulomatosis, Tolosa Hunt syndrome, Herpes zoster infection, Orbital phycomycosis, Pseudotumor due to aspergillus[32].

GRAVE'S DISEASE

Thyroid dysfunction is the most common cause of unilateral proptosis in adult hood. The proptosis frequently becomes bilateral in the course of the disease. Hyperthyroidism contributes to 90% of cases. Euthyroid & hypothyroid individuals form the rest 10%. Typical clinical signs accompany the disease. Proptosis with lid lag and lid retraction are very characteristic. Lid swelling, chemosis conjunctival hyperemia, lagophthalmos and impaired ocular motility can also be present. Defective vision and lacrimation caused by exposure keratitis may also be present. Impaired ocular motility is restrictive and mechanical in origin.Restriction is confirmed by "Forced duction test". Morphologically there is massive enlargement of the muscles due to interstitial edema. Individual muscle cells are normal.There will be associated inflammatory cell infiltration consisting of lymphocytes, macrophages, mast cells and plasma cells. These are manifestations of deranged immunologic system. Most patients have elevated levels of circulating thyroid stimulating immunoglobulins[86].

These are long acting thyroid stimulator and human thyroid stimulator (HTS) as well as easily demonstrable circulating antithyroid antibodies.

Systemic corticosteroids orbital radiotherapy and surgical decompression are the various methods adopted to treat Grave's exophthalmos.

ORBITAL FOREIGN BODIES

An orbital foreign body or a forgotten minor trauma to the orbit may result in proptosis, inflammation and infection of orbit. Insignificant and long forgotten, previous trauma may have introduced foreign material such as wood. Surgical exposure for diagnosis and drainage is needed. Culture of the purulent material for bacterial pathogens and especially for fungi is recommended.

VASCULAR LESIONS

Hemangioma

Capillary Hemangioma: It is the most common orbital lesion of infancy. Often not recognized during birth, it appears during the first month of life as an ill defined, compressible bluish mass that has a predilection for the upper nasal quadrant of the orbit. The proptosis increases in degree whenever the infant strains or cries. Capillary hemangioma of the eyelid is called a strawberry naevus. Capillary hemangioma grows with alarming rapidity during the first six months of life followed by spontaneous regression during the next four to five years. With involution it becomes more circumscribed[51]. Histologically these involutional changes represent a transition from a densely cellular hemangio endotheliomatous tumor into a hypocellular atrophic lesion that contains obliterated capillaries.Although natural involution occurs therapy with

irradiation or corticosteroids has been advocated to induce a more rapid regression.

Irradiation must be carefully monitored to avoid induction of cataract. The systemic side effects of steroids and the apparent rebound or reactivation of tumor growth upon stopping therapy must be kept in mind.

Cavernous hemangioma: It represents a developmental anomaly. Enlargement of the tumor is due to the sequential and progressive opening and dilatation of preexisting vessels and sinuses.Symptoms begin in early adulthood. A slowly progressive unilateral proptosis develops. Often the tumor is with in the muscle cone causing axial proptosis. It can also produce retinal striae by compressing the globe posteriorly. Visual loss, external ophthalmoplegia can also occur. Surgical removal of the mass is gratifying. Visual recovery is complete post operatively[39].

It is usually easily dissected free of other surrounding structures without hemorrhagic complications. Gross specimen typically has a honeycomb appearance. A connective tissue capsule surrounds widely dilated vascular spaces that are filled with red blood cells.

LYMPHANGIOMA

It is a rare congenital tumor of the orbit, becoming classically apparent in the early years of childhood. It tends to involve the superior orbit. It causes recurrent proptosis. Its slowly progressive relentless growth throughout

childhood, the lack of spontaneous regression and its unresponsiveness to therapy serves to distinguish it clinically from hemangioma.Bleeding into a lymphangioma causes rapid enlargement of the tumor and an increase in the proptosis and onset of pain. Such blood cysts are called “chocolate cysts”. Periodic enlargement of lymphangioma often accompanies upper respiratory tract infections causing recurrent proptosis[41].

ORBITAL VARICES

True incidence of orbital varices remains controversial. Many lymphangiomas have been mistakenly thought to be hemangiomas. Wright infact suggested that many lymphangiomas are in fact, congenital orbital varices[88].

Orbital varices are of two types

Type 1 Represents congenital weakness in the venous wall.

Type 2 Acquired weakness in the venous wall caused by an arterioveous shunt.

Signs and symptoms begin during the first five years of life. It continues to enlarge until the patient is 17 or 18 years of age after which there is little change. This also produces intermittent proptosis[53]. Proptosis results from straining, crying, valsalva maneouvre, placing the head in dependent position or compression of jugular veins. Proptosis is usually downwards, non pulsatile and with absent bruit. Enlargement of dilated veins in the eyelid may be present. Varices can be demonstrated by positive contrast venography.

ARTERIOVENOUS FISTULA

Orbital arteriovenous fistula is virtually non-existent. A tear in the internal carotid artery as it passes through the cavernous sinus produces a carotid cavernous fistula.

60% of cases are due to trauma. In 40% of cases it is spontaneous where the fistula is associated with a preexisting congenital defect in the wall of the intracavernous portion of the artery or an acquired degenerative vascular disease. The superior ophthalmic vein become the major route through which blood can escape from the engorged and distended cavernous sinus[51].

Patient complains of a swishing noise in the head synchronous with the pulse. A bruit is often heard on auscultation. Ophthalmoplegia, diplopia, proptosis, lid and orbital edema are also present. Dilated and tortuous conjunctival and episcleral veins are conspicuous. Unilateral pulsatile proptosis is evident which will proceed on to bilateral proptosis. Elevated episcleral venous pressure leads to ocular hypertension. The associated glaucoma is difficult to treat.A high index of suspicion and careful neuroradiologic studies will usually lead to the correct diagnosis. Common carotid ligation on the affected side will cause regression of all the findings. Dural cavernous sinus fistula is a low flow, low pressure shunt unlike CCF.

TUMORS

MALIGNANT HEMANGIOENDOTHELIOMA

Malignant tumor of the endothelial cells of the blood vessels. These tumors are uncommon. These tumors are infiltrative and locally aggressive and have a high metastatic potential.

HEMANGIOPERICYTOMA

This is a tumor of the pericyte which occupies a position outside of the endothelial cells but in close apposition to them in capillaries and postcapillary venular channels. Usually presents with proptosis of 6 months to 2 years duration. Tumor is usually situated within or outside the muscle cone and usually does not involve bone at the onset. Eyelid swelling, diplopia, a palpable mass in 60% of patients are present.

LACRIMAL GLAND TUMORS

Benign mixed tumor:

It is the most common epithelial neoplasm that affects the lacrimal gland[92].

It usually occurs in males between 20 and 50 years of age presenting with tearing and a painless mass within the lacrimal fossa. The mass may be asymptomatic for years. Attention is directed to the tumor by the eccentric proptosis and diplopia.

Because of the frequency of recurrences, which are often multinodular, the tumor is excised widely, often including nearby periorbita or bone. If a biopsy is necessary, it should be performed with great care. Histologically it is a pleomorphic adenoma.

MALIGNANCIES

Malignant neoplasms account for 50% of the epithelial tumors of the lacrimal gland. Unlike benign mixed tumor it produces rapid enlargement of the gland during a short period. Eccentric proptosis-downwards and inwards, pain and tenderness to palpation further support the diagnosis. X-ray reveals enlargement of the lacrimal fossa, increased bone density or bone destruction. Malignancies of the gland are almost always lethal, regardless of therapy unlike mixed tumor. Occasionaly the sudden increase in size of a formerly insignificant lacrimal fossa heralds malignant transformation within a benign mixed tumor. Adenoid cystic carcinoma is the most common malignancy of the lacrimal gland. Adenoid cystic carcinoma and benign mixed tumor tend to occur in the same age groups[71].

Adenocarcinoma, squamous cell carcinoma, mucoepidermoid, sarcoma can also occur within the lacrimal gland but very rare. Orbital exenteration carried out at the initial surgery lessens the futility of treatment methods.

NEUROGENIC LESIONS

NEUROFIBROMATOSIS

Ocular manifestation occur in the first decade of life. Although hereditary, it is transmitted in an autosomal dominant fashion. Orbital involvement is invariably unilateral. One characteristic finding is developmental absence of orbital bones posteriorly. These defects usually involve the sphenoid bone and orbital roof posteriorly. Harkin and Reed have classified involvement in neurofibromatosis into 3 principal patterns.

1. Central neurofibromatosis with gliomas, ependymomas, meningiomas & schwannomas of the neuraxis.
2. Peripheral neurofibromatosis with neurofibromas of the skin, plexiform type tumors and sparing of CNS and the viscera.
3. Visceral neurofibromatosis with neurofibromas schwannomas and ganglioneuromas occurring along the Gastero Intestinal tract[39].

PERIPHERAL NERVE TUMORS

SCHWANNOMA (NEURILEMMOMA)

These slow growing tumors are usually located behind the globe. They develop in the middle age usually in isolation from neurofibromatosis and may produce considerable proptosis upto 10 mm. Peripheral nerves contain 3 cell types that may participate in the formation of tumors-the schwann cell, the

endoneural cell and the perineural cell. These tumors occur in the orbit either as part of neurofibromatosis or totally independently. Schwannomas compress the nerve of origin and hence cause pain on retropulsion. Neurofibroma, plexiform neurofibroma, malignant schwannoma, neuroepithelioma, orbital melanoma are other tumors that can affect the peripheral nerves.

MENINGIOMAS

Can be primary or secondary

Primary: Orginates from the intraorbital optic nerve sheath.

Secondary: Originates along the sphenoid wing or in the basofrontal region and subsequently invade the orbit.

Primary

Two varieties of primary intraorbital meningioma infrequently occur.

1. Those that derive from ectopic arachnoid cell within the muscle cone unattached to the optic nerve or orbital wall.
2. Those that appear to derive from the periorbita of the orbital walls.

   Meningiomas situated along the optic nerve especially behind the globe will present as proptosis and visual loss.Meningiomas situated more posteriorly in the orbit around the foramen will present as proptosis much later.In young females there is predilection for the right orbit. Papilledema, optic atrophy, scotomas are common features.Optico

ciliary shunt vessels can open up on the optic nerve head to direct retinal venous blood around the obstructed optic nerve towards the choroid.

Secondary

Meningiomas which secondarily involve the orbit arise either from the wings of the sphenoid or in the basofrontal area near the tuberculum sellae. Reactive hyperostosis seen in x-ray is very characteristic. Causes proptosis, visual loss, ophthalmoplegia and nasal hemianopia.

GLIOMA

Optic nerve gliomas are uncommon but they are more common than meningiomas. The peak incidence is from 2-6 years of age. Females are slightly more frequently affected[29]. Clinical presentations fall into 2 patterns depending on whether the tumour is largely orbital or intracranial. Loss of vision is the most common initial symptom in intracranial gliomas. Proptosis is an early feature of intraorbital gliomas. It is usually very mild rarely exceeding 3mm.Proptosis is axial, non pulsatile and irreducible. Pain is unusual. Tumor is not palpable. Movements are mechanically restricted.

Ophthalmoscopy reveals primary optic atrophy, CRVO is a rare complication.

Intracranial gliomas disturb hypothalamic and pituitary function and produce symptoms of increased intracranial pressure. Proptosis is also present if there is orbital involvement.

RHABDOMYOSARCOMA

It is the most common primary malignant orbital tumor of childhood.

Produces a rapidly progressing unilateral proptosis of sudden onset in a child, seven to eight years of age.Although the proptosis, lid swelling and chemosis may be marked, the symptoms associated with this rapidly evolving and often horrifying clinical picture may be disproportionately meagre.

It is a highly malignant neoplasm of pleuripotential embryonic mesoderm[31].

FIBROOSSEOUS LESIONS

OSTEOMA

Osteoma is a common benign tumor of the paranasal sinuses. It usually arises within the frontal sinus. It is less frequent in the ethomoidal and maxillary sinuses and rarely seen in sphenoidal sinus. They grow very slowly and is usually asymptomatic for long periods of time, frequently being discovered as an incidental finding. Headache, facial pain or swelling are the usual complaints.Nasal obstruction or discharge may be caused by an ethmoidal osteoma. Frontal and ethmoidal osteomas are most likely to encroach on the orbital space. When palpable it is firm hard, nontender, and noncompressibel. Asymptomatic osteomas, especially in elderly require no therapy.

Encroachment into the orbit requires surgery. Histologically it consists of dense intracommunicating bony lamellae.

FIBROUS DYSPLASIA

It is a nonneoplastic disorder of the childhood that frequently involve the orbital bones. The frontal, sphenoidal and ethmoidal bones are most commonly involved.

Thickening of these bones decrease the orbital volume and characteristically leads to a slowly progressive unilateral eccentric proptosis. Facial asymmetry will be conspicuous. The condition becomes inactive after puberty. Surgical intervention may become necessary, especially when there is optic nerve compression producing visual loss.Histologically poorly calcified, primitive islands, of bone are surrounded by a highly cellular stroma that is composed of benign fibroblasts[3].

ANEURYSMAL BONE CYST

They are less common in the orbit. Expanding lesion of the bone involving the orbit and producing proptosis. Usually involves the roof of the orbit.

X-ray reveals eccentric, ballooned out area covered by a thin layer of bone cortex. The ideal goal of treatment is complete excision. There is some evidence that the tumor may not recur if completely excised.

Other lesions like ossifying fibroma, osteoblastoma, osteogenic sarcoma can also produce proptosis.

SECONDARY ORBITAL INVOLVEMENT

FROM THE GLOBE

Retinoblastoma

Presence of orbital extension indicates a fatal outcome. Orbital extension is associated with retinoblastomas that involve the choroid and optic nerve extensively. Once in the orbit, tumor grows luxuriantly causing rapidly advancing proptosis or extrusion of implant.

Malignat Melanoma

Proptosis may result from an enlarging melanomatous epibulbar nodule. There is usually a prolonged delay in diagnosis, often related to the presence of a flat or diffuse intraocular melanoma[19].

Medulloepithelioma

Embryonal tumor originating from primitive optic vesicle neuroepithelium in the ciliary body region in children or exceptionally arise from persistent embryonal medullary epithelium near the optic nerve head producing proptosis.

FROM THE LIDS

Malignant melanoma

Not so common. Invasion of the orbit occurs as a part of recurrence and frequently necessitates exenteration.

Sebaceous carcinoma

Produces displacement of eye rather than proptosis due to anterior[19] location.

Basal cell carcinoma

This also produces proptosis rarely. Extension into the orbit is often accompanied by bony invasion at the orbital rim[7].

FROM THE PARANASAL SINUSES

Carcinomas spread from the four surrounding sinuses into the orbit with relative ease because of the thin bony walls separating them from the orbit and also because of the various fissures and foramem in them. 80% of malignant tumors of sinuses are carcinomas and 20% are sarcomas.

Proptosis will be eccentric and the direction of it will be opposite the site from where the tumor enters the orbit. Diplopia and visual loss can also occur. Ethmoidal carcinoma is more common in females. Maxillary carcinoma is more common in males. There is a male predominance in the incidence of carcinomas invading the orbit from the nose.

Epiphora and pain occur early in tumors arising from ethmoid and maxillary sinuses. Usually the sinus tumors are discovered late and by the time they have invaded the orbit only extensive and radical surgery can cure them.

FROM NOSE AND OROPHARYNX

Carcinoma

Squamous cell carcinoma of the nasopharynx is the commonest malignancy to invade the orbit from the nose and oropharynx. They cause proptosis, epiphora, ophthalmoplegia, multiple cranial nerve palsies, Horner's syndrome.

Melanoma

Melanonas of the nasal cavity, maxillary antrum and ethmoidal sinus can invade the orbit. Usually patients die within five years due to local spread or metastasis.

Angiofibroma

It is a benign vascular tumor yet with dangerous infiltrating capacity by local spread. Usually occurs in young adults and can cause proptosis, visual loss.

Secondary involvement of the orbit can occur also from lacrimal sac and cranial cavity lesions.

METASTASIS

Neuroblastoma

It is one of the most common childhood tumors. CNS tumors and leukemia are more common in the pediatric age group.

It is a neoplasm of embryonic neuroblastic tissue. It may arise anywhere such tissue is normally found. There is one report of a neuroblastoma having arisen within the orbit.

Apart from proptosis, flushing, diarrhea and hypertension are seen in some patients with secreting tumors. In the orbit the metastasis has a tendency to lodge in the zygomatic bone. Ecchymosis of lids is also present.

Leukemia

In over 75% of patients with leukemia the eye and adnexa are invaded by leukemic cells, usually by lymphoblastic cells. The acute leukemias are more likely to cause orbital metastasis and lymphoblastic ones more so than myelogenous leukemias. Orbital involvement occurs in 2% of patients. In adults lung carcinoma in male and breast carcinoma in females are the most common primary lesions causing orbital metastasis.

Breast, Lung, Kidney, Testicle, pancreas, Colon, Rectum, Stomach, Thyroid, Ileum are other primary sites form which metastasis occurs.

INVESTIGATION OF PROPTOSIS

ORBITAL LESIONS

It may be classified as

- Congenital and developmental anomalies
- Traumatic lesions
- Inflammatory and infectious lesions
- Neoplastic lesions

CLINICAL EVALUATION OF PROPTOSIS

This is based on the following:

A. History

B. Clinical examination

C. Investigation

D. Differential diagnosis

E. Choice of therapeutic regimen

HISTORY

The following points are relevant:

1. Usual complaints of orbital disease are -

   proptosis/pain/diplopia/defective-vision/defective field of vision

2. Mode of onset may be acute/chronic/congenital/acquired

3. Mode of progression - may be stationary/gradually progressive or rapidly progressive Remissions and exacerbations may also occur.
4. Diurnal variation-Painful proptosis in the early mornings which subsides by evening indicates orbital inflammatory diseases like pseudo tumor, thyroid orbitopathy etc.
5. History of injury to eye or head injury
6. Systemic complaints:

a. Thyroid related complaints like, increased appetite with loss of weight, Palpitations and chest pain, Increased/decreased appetite, Hyperactivity/lethargy, Skin related problems, Swelling in the neck region, tremors in the extremities, menorrhagia / Amenorrhoea in females.
b. History of fever and frequent upper respiratory infections are important in case of leukemias, lymphangiomas, orbital inflammations and sinusitis.
c. History of epistaxis and abnormal bleeding from other sites of body
d. History of loss of weight and loss of appetite may be suggestive of systemic malignancy
e. History of any other swellings in the body as in the case of neurofibromatosis, haemangioma, parasitic cysts.

f. Hypertersion/diabetes/cardiovascular/renal/respiratory/gastrointestinal/ skeletal/neurological history

CLINICAL EXAMINATION

Systemic Examination[45]

This should include:

1. Vital signs monitoring: blood pressure, pulse rate, respiratory rate and temperature recording
2. Lymphadenopathy which may be preauricular/cervical/generalized
3. Hepatosplenomegaly
4. Any other mass palpable in other sites of body
5. Thyroid swelling
6. Finger tremors/clubbing/cyanosis
7. Nasopharyngeal examination/Dental examination
8. Any musculoskeletal disorders/Dermatological examination

   Dry scaly skin in thyroid Café-au-lait spots-neurofibromatosis
9. CNS examination
10. CVS/Resp/GIT Examination

LOCAL EXAMINATION

General

1. Visual status and refraction: Patient may be hypermetropic because retina is pushed forwards, or myopic if there is a pseudoproptosis.
2. Color vision
3. central field charting
4. Diplopia and Hess charting
5. Squint evaluation/Forced duction test
6. Examination of anterior segment/slit lamp biomicroscopy
7. Pupillary reactions-Direct/consensual
8. Intraocular pressure should be done preferably with applanation tonometry. It increases in orbital inflammatory diseases. Differential intraocular pressure recording should be done in various gazes.
9. Examination of posterior segment by indirect/direct/ ophthalmoscope

Fundus examination to rule out papilloedema/papillitis/optic atrophy/opticociliary shunt/vascular anomalies/retinal striae or folds, choroidal folds, exudative retinal detachments etc .

Specific examination

This should be carried out for:

1. Facial symmetry.

2. Position of eye brows/eye lids-lid retraction/bogginess of lids/lid lag on downgaze.,
3. Examination of ptosis/measurement of levator function,
4. Extraocular motility/defective convergence,
5. Various thyroid orbitopathy signs

PROPTOSIS

It may be axial or eccentric depending upon direction. It may be unilateral or bilateral.

- Down and out proptosis occurs in ethmoidal mucocles.
- Down and in proptosis occurs in lacrimal gland tumors
- Up wards in maxillary involvement.
- Downwards in subperiosteal hematomas

ON INSPECTION

Naffziger's sign: While looking tangentially over the forehead, the palpebrae of the proptosed eye is seen first.

- Other points to be noted are fullness/mass lesion in the orbit
- Any visible pulsation/visible engorged vessels,
- Lagophthalmos with cornel exposure.

- Conjunctival congestion at recti muscle insertion indicates thyroid orbitopathy while diffuse congestion of conjunctiva indicates vascular anomaly.

ON PALPATION

Palpation of orbital rim for any irregularity, mass lesion, finger insinuation between globe and orbital bones is essential.

Size, shape, surface, skin over swelling, consistency, signs of inflammation, tenderness, reducibility, margins ,motility etc., details of the mass lesion must be noted.

Variation of proptosis with alteration in postures are like valsalva maneuver/bending down the head, should be looked for.

Other features to be noted are

- Resistance to retropulsion,
- Pulsations over swelling/over the eye ball,
- Infra orbital/supraorbital anaesthesia, Corneal anaesthesia

ON AUSCULTATION

For the presence of bruit over the mass lesion/over the eye ball/over the temporal vessels. It is positive for A-V malfomations.

INVESTIGATIONS

Can be divided into

- **Lab investigations**
- **Non invasive techniques**
- **Invasive techniques**

LAB INVESTIGATIONS[45]

Total Count**:** in orb leukemia, TB, syphilis

Differential Count **:** Eosinophils raised in Toxocariasis , Echinococcosis, Parasitic cysts, Angioneurotic oedema

ESR: TB - of orbit ,Tuberculoma, Multiple myeloma

Mantoux Test **:** Positive in proptosis of TB etiology

Peripheral Smear**:** Leukemia (immature erythroid and myeloid precursors)

Blood sugar**:** Mucormycosis

VDRL**:** syphilitic etiology

Thyroid function tests**:** Estimation of T3,T4,TSH, radioactive Iodine uptake tests.

Skin tests**:** Casoni's test for hydatid cysts

Kveim's test for sarcoidosis

LDH assay: 32 uptake test confirms a RB

NON INVASIVE

1. Plastic Ruler (I.ueddel's Method)

2. Exophthalmometry[12]

Measurement of the distance between the apex of cornea and a bony point on the deepest part of the lateral margin of the orbit with the eyes looking straight ahead.

Types

Hertel's Exophthalmometer, Zehender, Gormaz, Davanger's ,

Radiographic and stereographic methods

Values

Normal distance<20mm. if> 21mm it is abnormal - absolute

Difference of >2mm between two eyes - relative

3. Plain X-Ray orbit

Summary of radiographic projections

Projection	**Structures**	**Pathology**
Water's view (OM view)-PA view	Orbital floor (ant.2/3) and maxillary sinus	Maxillary sinus disease
Caldwell's view (OF view)-PA view	Frontal and eth sinus, sph.bone, SOF	Meningeoma of sph.sinus
Lateral view	Sella tursica, sinus air	Pituitary disease, frontal

	fluid level	sinus disease
Basal view	Sph & eth sinus, lateral wall of orbit	Sinus disease
Optic foramen view or rhese view or oblique apical view	Optic foramen	Apex tumours, optic nerve tumours
Towns view-AP view	Inferior orbital fissure	

CHANGES IN PLAIN X-RAYS

X-RAY - ORBIT

Enlargement of orbit:

Localized-any longstanding LG tumor, Diffuse-intraconal benign lesions.

Diminution of orbit:

Cranio synostosis , Mucocele of adjacent tissues, after therapeutic irradiation.

Bone destruction:

- Congenital bony dehiscense

- Localized bony destruction with a clearcut margin- multiple mylom, Dermiod, Epidermiod, Histeocytosis
- Diffuse irregular osteolysis due to malignancy - Primary, Metastatic, Invasion from adjacent structures

Enlargement of SOF:

Meningioma, CCF, Infra clinoid aneurysm, Extra sella extension of pituitary tumour.

Enlargement of optic foramen:[84]

Normal diameter-4 to 6mm, Greater than 7mm-abnormal, normal difference between two sides greater than 1mm - abnormal

- Uniform widening-optic nerve glioma
- Asymmetrical widening of margin – meningioma
- Total destruction of outline – malignant invasion from PNS

Soft tissue changes:

- Mucoecle, clouding of PNS in infection, neoplasms, hemorrhage's

X RAY SKULL:

- Pituitary tumour - Sella enlargement, Beaten metal appearance, Erosion of clinoid processes.
- Craniopharyngeoma - Supra sellar calcification
- Areas of calcification - Hydatid cyst, Tuberculoma, Aneurysms

X RAY SINUSES :

Mucocele, Carcinoma, fungal involvement .

X RAY CHEST:

TB, Sarcoidosis, Rhabdomyosarcoma, Secondaries from Breast carcinoma

X RAY PELVIS:

Prostatic carcinoma

ORBITONOMETRY(Peizometry):

It measures the compressibility of orbital contents an differentiates between solid and cystic lesions.

HOLOGRAPHY-3d images is produced in a single exposure by a special photographic process.

ULTRASONOGRAPHY :

- A - SCAN – Vascularity, Growth
- B - SCAN

Confirms the presence of tumour, detects calcification, dimensions of tumour, determines if solid/cystic[18]

Thyroid ophthalmopathy -Enlargement of EOM, RB fat

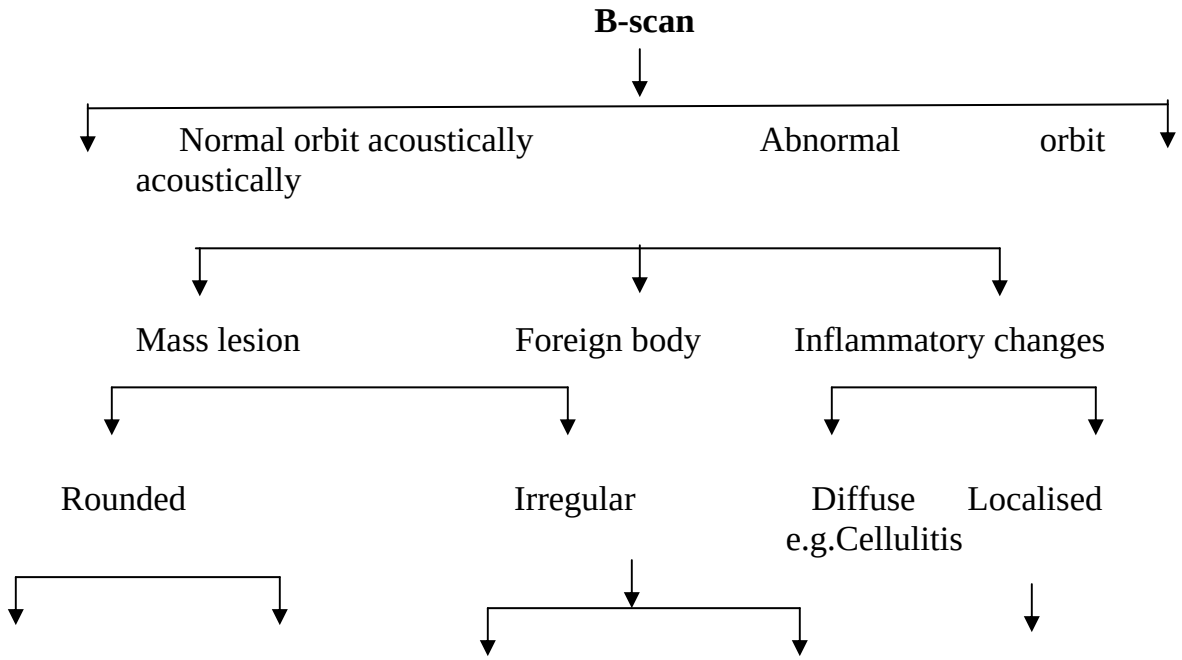
Pseudo tumour - Oedema of normal orbital structures, Irregular mass lesion

LG tumour - Well demarcated contour

Rhabdomyosarcoma, Glioma, Dermoid, AV fistula are also delineated

Schematic Flow Chart of Orbital Diagnosis

Unknown Orbit



COMPUTED TOMOGRAPHY[58]

- Diagnostic as it detects very small lesions seen as bright calcification.

- Endocrine exophthalmos-Thickening of EOM confined to the belly
- Myositis- EOM enlargement is from insertion to origin
- Pseudo tumour-Enlargement of EOM due to infiltrates
- Optic nerve glioma-Solitary uniform enlargement of optic nerve
- Meningioma -Thickening of the optic nerve with a mass lesion[52].
- Bacterial orbital cellulites - Mucosal thickening, sinus opacification
- Orbital varices - vascular marking, enlargement of orbit

MAGNETIC RESONANCE IMAGING

Used in

- RB - Presence of optic nerve involvement , Presence of ectopic intra cranial RB, however CT is superior as it detects calcification better
- Orbital cellulites - Linear streaking of normal fat shadows in T2 weighted images
- Optic nerve glioma, Optic nerve sheath meningioma, Thyroid sheath meningioma, Thyriod orbitopathy, Rhabdomyo sarcoma, LG tumour, Capillary hemangioma, Cavernous hemangioma

Contra indication of MRI :

Ferro magetic FB, Pace marker, Aneurysm clips

INVASIVE TECHNIQUES

Arteriography[16]

- Demonstrates both intra cranial and orbital SOL, aneurysm,CCF
- 60% urograffin used
- Localised compression/dilation/displacement of vessel in tumours
- Circulatory abnormality in vascular lesion
- Carotid angiography

Venography

- Orbital veins are rendered visible by injecting contrast medium through angular vein, frotal vein or super orbital vein.
- Both facial veins must be compressed at the time of injection to prevent escape of contrast medium over the scalp by placing a tight rubber band around the hair line.
- Both SO veins fill simultaneously allowing direct comparison of abnormal and normal side.
- Subtraction technique studies give clearer outline of the veins

1. AP view SOV is seen as a parallelogram.

Extraconal lesion-closing of parallelogram-angles are acute.

Intraconal lesion- opening of parallelogram-angles are obtuse.

(Exception is an extraconal lesion on the medial side of the orbit)

2. If vascular lesion it can be demonstrated by filling up of veins e.g orbital varices

3. Compression of SOV at the apex by tumours leads to obstruction of SOV

4. Endocrine exophthalmos-minor displacement of veins

Radio isotope scanning (orbitography):

- Scan the different conc.of IV administered radioactive isotopes.
- Early localization of radioactivity-benign tumour.
- Late development of radioactivity-malignant tumour.

FNAC:

- FNAC is mainly indicated for secondary tumours.
- Disadvantage in primary tumours- seedling of tumour cells

Bleeding due to increased vascularity

SURGICAL MANAGEMENT OF PROPTOSIS

Indication for orbital surgery:[75]

1. Open sky biopsy of orbital tumours / granulomas.
2. Complete excision of benign tumours / parasitic cysts.
3. Debulking of large benign tumour / excise malignant tumours before radiotherapy.
4. removal of orbital foreign body is usually not indicated unless vision is threatened.
5. Orbital fracture reduction if there is intractable diplopia.
6. Orbital wall decompression in cases of endocrinal exophthalmos.

Following questions summarise the tips in formulating a surgical plan.

What is the most likely diagrosis?

Is excisional biopsy or incisional biopsy preferred?

What is the who best surgical approach?

TYPES OF ORBITAL SURGERY

LINE	SPECIAL	RARE
Lateral orbitotomy	trans ethmoidal orbitotomy	Exenteration
Inf, med sup orbitotomy	Trans conjunctival	
Orbital wall decompression	Cranial	

Surgery	Nature of the lesion	Example	Possible
ANTERIOR ORBITOMY	Anterior localized Anterior Diffuse Posterior Diffuse Lacrimalglandlesions	Dermoid cyst Lymphoma, abscess Pseudotumour, abscess, metastatic lesions Mg.Carcinoma	Excision Biopsy drainage Biopsy drainage Biopsy
MEDIAL ORBITOTOMY	Medial, anterior localized Medial, posterior localized Medial, anterior diffuse Optic nerve lesions	Varix, hemangioma Cavernous hemangioma, pseudotumor Abscess, Rhabdomyosarcoma Glioma, Meningioma	Excision, Biopsy Excision, Biopsy Drainage Biopsy Biopsy, Excision
LATERAL ORBITOTOMY	Posterior localized Posterior diffuse	Meningioma, Hemangioma Metastatic, Pseudotumors	Biopsy, Excision Biopsy, Decompression Drainage
EXTENDED LATERAL ORBITIOTOMY	Localised posterior apical Lacrimal gland fossa lesions	Meningioma Benign mixed tumour	Excision, Biopsy Excision with adjacent tissue
EXENTERATION	Extensive malignant orbital lesions	Lacrimalgland Carcinoma,	Excision with orbital contents.

Usual guidelines for orbital tumour surgeries:

- Communicate with the pathologist before the surgery
- Good illumination
- Sufficient magnification
- Blunt dissection within orbit
- Palpate any mass as dissection proceeds
- Handle any pathological specimen very carefully avoid crushing.

COMPLICATIONS OF ORBITAL SURGERY[69]

Vascular: CRVO, optic nerve ischaemia & atrophy, vitreous and orbital haemorrhage.

Neural/muscular: Corneal anaesthesia, internal ophthalmoplegia muscle paresis, ptosis, lateral rectus adhesion.

Inflammatory: Exacerbation of pseudotumor

Others: Enophthalmos, xerophthalmia, Retinal tears & Detachment.

PART – II

REVIEW OF LITERATURE

1. Henderson's orbital tumour series from the Mayo clinic and with Rootman's series from British Columbia, Fredrick A, Jokabie , Daniel M, Albert , Dimitri T Azar , Evangelos S. Gragoudas which source the age distribution form birth to 92 years median age of 47 years most common involvement steadily increases form the third through sixth decade.
2. Fredrick A, Jokabie , Daniel M, Albert , Dimitri T Azar , Evangelos S. Gragoudas which showed 52% of males and 48% of females and Shilvas orbital survey study showed 57% of male predominance.
3. The side involved is predominantly right side in Mombaerts I, Goldschmeding R, Schlingemann RO, et al. What is orbital pseudo tumors Surv Ophthalmol. 1996; 41: 66-78. which showed right sided predominance for no specific reason known.
4. Henderson J: Orbital Tumors 3rd ed. New York, Brain C, Decker, 1994, pp 43-51. Rootman J: Diseases of the orbit. Philadelphian, JB Lippincott, 1988, pp 1-612. which says more than 50% disease of orbit presents eccentrically.
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biopsies. Arch Ophthalmol 102: 1606 – 1611, 1984. which shows 50% of cases of gradual onset of proptosis.

6. Newton TH, Bilaniuk LT, eds Radiology of the eye & orbit (Modern neuro radiology) volume 4 New York Ravan; 1990 describes standard instruments pick out lesion of 3-5mm more earlier.
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14. Wilson MW, Buggage RR, Grosniklaus HE: Orbital lesions in the South eastern United States. Orbit 15; 17- 24. 1996.
15. Dallow RL: Momuse KJ, Seber AL, Wray SH; Comparison of ultrasonography, computerized tomography and radiography, ophthalmology 85; 1218, 1978.
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AIM OF THE STUDY

1. Demographic study of unilateral proptosis
2. Incidence of various causes of unilateral proptosis
3. Effect on vision and visual recovery following treatment
4. Cure rate possible in various etiological causes.
5. Analyzing the significance of clinical symptoms signs and directing appropriate laboratory imaging investigation
6. Influence of patients age on diagnosis consideration.

MATERIALS AND METHODS

All the patients were selected form the orbit clinic of the Department of Ophthalmology. Govt. Rajaji Hospital, Madurai

Each patient was thoroughly examined in the same department. After recording the detailed history including past history , general and ocular examination were carried out.

Exophthalmometry was done for all cases. Detailed examination of the proptosis was done to know whether it is axial or eccentric, its reducibility, pulsatility, tenderness, thrill, bruit, presence of visible mass and change associated with cough impulse and valsalva maneouvre.

Investigations like total count, differential count, urine sugar, peripheral smear, VDRL and mantoux test, were done in our department laboratory. X-rays, CT scan and MRI were done in the radiology department.T_3, T_4 and TSH estimation were done in the endocrinology department. Doppler study was done for two patients soutside.

Lateral and medial orbitotomies were done for appropriate lesions.

Cases were followed up in the eye department. All the findings were entered in a proforma for further analysis.

Cases of trauma that presented with unilateral protopsis and pseudoproptosis cases were excluded from the study.

RESULTS AND OBSERVATION

During the period of study for 2yrs February 2004 to March 2006 the total number of patients with unilateral proptosis examined were sixty.

The number of males was 29. The numbers of females were 31.

No of children < 10years were four.

TABLE I

AGE AND DISTRIBUTION

No	Age in years	Cases	Percentage
1.	0-10	4	7%
2.	11-20	5	8%
3.	21-30	6	10%
4.	31-40	9	15%
5.	41-50	11	18%
6.	51-60	10	16%
7.	61-70	9	15%
8.	71-80	4	7%
9.	>80	2	4%

II. THE SIDE INVOLVED WERE

Right eye : 31 (51.6%)

Left eye : 29 (48.14%)

III. THE TYPE OF PROPTOSIS ENCOUNTERED

Axial : 28 (46.48%)

Eccentric : 32 (53.12%)

IV. PAINFUL PROPTOSIS WAS SEEN IN 25 (41.5%) PATIENTS

V. THE ONSET OF PROPTOSIS WAS

Sudden in days to weeks 25 (41.5%) cases

Gradual in Months to years 30(49.8%) cases

Since birth in 2 (3.32%) cases

VI. AMOUNT OF PROPTOSIS

Table II

NO.	PROPTOSIS	NUMBER	PERCENTAGE
1.	0-2 mm	5	8.3%
2.	3-5 mm	30	49.8%
3.	6-10 mm	18	29.66%
4.	>10 mm	2	3.3%

In 5 children proptosis could not be measured.

VII. DIPLOPIA

Was observed in 6 cases. (10%). It totally subsided in 4 patients.

VIII. FUNDUS EXAMINATION

Primary optic atrophy was present in 7(11.62%) patients.

2(3.3%) patients with orbital varices presented with dialted, tortuous retinal veins.

Rest of the patients had a normal fundus.

IX. PRIMARY AND SECONDARY ORBITAL LESION

Of the 60 cases, 44(73%) patients presented with primary orbital pathology while in the remaining 16(27%) patients the orbit was involved secondary to a systemic cause.

Table III

	Cases	**Percentage**
Primary orbital involvement	44	72.5
Secondary orbital involvement	16	27.5

X. CONFIRMATION OF CASES

In 18(30%) cases diagnosis was confirmed by histopathological examination. In 2 cases of orbital varcies diagnosis was confirmed by doppler studies.

In the remaining 40(66.4%) cases diagnosis was arrived at after ratioimaging studies, therapeutic trial and observation.

In a 3 years old child with proptosis of left eye due to orbital secondaries from adrenal neuroblastoma the right eye presented with orbital deposits but without proptosis.

XI. INCIDENCE

No of cases referred to orbit clinic – 108

Bilateral proptosis - 48 (44.4%)

Unilateral proptosis - 60 (55.55%)

The ratio of bilateral and unilateral proptosis is 1:5 .

XII. AETIOLOGICAL CLASSIFICATION

Table IV

Aetiology	No. of cases	Percentage
Congenital	2	3.32%
Inflammatory	23	38.18%
Grave's disease	12	25%
Vascular	2	3.32%
Tumour	7	11.62%
Sinuslesion	4	6.64%
Metastasis	4	6.64%
Orbital cellulites	3	4.98%
Miscellaneous	3	4.98%

XIII. TUMOURS

Table V

Primary	Cases	Percentage
Neruogenic	2	3.32%
Lymphoma	1	1.66%
Dermoid	1	1.66%
Angio myofibrosarcoma	1	1.66%
Lacrimal gland	1	1.66%
Rhabdo myosarcoma	1	1.66%

Metastasis	Cases	Percentage
	4	6.64%

XIV. SINUS LESIONS

Table VI

	Cases	Percentage
Mucocele	2	3.44%
Mucormycosis	1	1.66%
Ca. maxilla	1	1.66%

Inflammation and neoplasms constitute more than 55% of the cause for unilateral proptosis in children under 15 years of age.

XV. TREATMENT

26 patients were treated medically with drugs alone – both oral and parentral drugs.

SCHEDULE

- Tablet predinisolene acetate 1mg/kg body weight was given for 1week
- 10mg was tapered per week.
- 40mg /kg body weight was maintained for 3weeks.
- Further 10mg/kg was tapered for 3 more weeks.
- 5mg for 1week was given and patient was weaned of drug.
- Frequent blood sugar level fasting and postprandial were taken.
- Adverse drug reactions were monitored
- Exophthalmometry reading repeated in each visit.

Rapid reduction of systemic steroids may cause recurrence of inflammatory signs and symptoms. As per American academy of ophthalmology orbit , eyelids and lacrimal system section 7 2001-2002. Pg.54.

- 8 cases were treated by medical and surgical procedures like incision and drainage, biopsy , fine needle aspiration were done to investigate the cause.

- 3 cases of orbital cellulites were treated with injection cefotaxime 1gram intravenous two times a day. Topical antibiotic ciprofloxacin eye drops 6 times per day, analgesics and antiinflammatory, with surgical drainage procedure.
- Enucleation was done for two cases of optic nerve glioma.
- Exenteration was done for 2 cases of orbital and sinonasal mucomycosis and cancer sinus maxilla extending in to orbit.
- Orbitotomy and tumor removal was done conjointly with neurosurgeons for 7cases, Lymphosarcoma orbit (1), malignant lacrimal gland tumor(1) , angiomyofibrosarcoma (1), orbital varcies (2), lateral dermoid (1).
- Lateral orbitotomy and radiotherapy was given for a cases of Rhabdomyosarcoma .
- Endonasal sinus surgery was done for 2 patients with mucocele sinus extending in to orbit.
- Chemotherapy was given for two patients who presented with metastasis.
- 10 patients were just put under observation.

XV. TREATMENT SCHEDULE

Table VII

Treatment	Cases	No of cases	Percentage
Medical	I(16) ,T (6)	22	36.52%
Medical & Surgical	OC (3),MIS (3), I(5)	11	18.26%
Observation	C(2), T(2), I(2), M(2)	12	19.92%
Lateral orbitotomy	V(2), T(4),	6	9.96%
Orbitotomy + Radiotherapy	T(1)	1	1.66%
Chemo therapy	M(2)	2	3.32%
Sinus surgery	S(2)	2	3.32%
Enucleation	T(2)	2	3.32%
Exenteration	S(2)	2	3.32%

Surgery was carried out in patients

- Enucleation was done in 2 patients
- Exenteration was done in 2 patients
- Surgery followed by radiation was given to 1 patients
- Chemotherapy was given to 2 patients.

XVI. EVALUATION OF PROGNOSIS

Table VIII

Progress	No. of cases	Percentage
Total resolution	18	29.88%
Observation	10	16.6%
Partly resolved	12	19.92%
Enucleation	2	3.32%
Exenteration	2	3.32%
Lost followup	5	8.3%
Irregular treatment	9	14.94%
Death	2	3.32%

- Total resolution of proptosis was achieved in 18 cases.
- Two cases landed up with enucleation of the involved eye.
- Two cases needed exenteration of the orbit for total amelioration.
- Five patients absconded while under treatment.
- In twelve cases the condition had party resolved when this study ended.
- Ten patients were kept under observation without active intervention.
- Ten patients were taking irregular treatment with no regular check up.
- Two patients died during the period of study.

XVII. VISUAL PROGNOSIS

The visual acuity recorded at the end of treatment with complete resolution or at the end of this study are grouped as follows

VISUAL PROGNOSIS

TABLE - IX

Visual acuity	**No of cases**	
	Initial	**Final**
6/6 to 6/12	15 (24.9%)	30 (49.8%)
6/18 to 6/60	16 (26.56%)	9 (14.94%)
<6/60	15 (24.9%)	7(11.62%)
No PL	11(18.26%)	11(18.26%)
Cannot assess	3 (4.98%)	3 (4.98%)

DISCUSSION

The age distribution range from 0-82 years median age of 45 years most common involvement being in the fourth decade (18%) which is consistent with Henderson's orbital tumour series from the Mayo clinic and with Rootman's series from British Columbia, Fredrick A, Jokabie , Daniel M, Albert , Dimitri T Azar , Evangelos S. Gragoudas which source the age distribution form birth to 92 years median age of 47 years most common involvement steadily increases form the third through sixth decade.

Predominance was found in males (51.46%0 followed female 48.14% which is consistent with study of Fredrick A, Jokabie , Daniel M, Albert , Dimitri T Azar , Evangelos S. Gragoudas which showed 52% of males and 48 of females and Shilvas orbital survey study showed 57% of male predominance.

The side involved is predominantly right side consistent with Mombaerts I, Goldschmeding R, Schlingemann RO, et al. What is orbital pseudo tumors Surv Ophthalmol. 1996; 41: 66-78. Which showed right sided predominance for no specific reason Known.

Eccentric is more common than axial proptosis which is comparable with Henderson J: Orbital Tumors 3rd ed. New York, Brain C, Decker, 1994, pp 43-51. Rootman J: Diseases of the orbit. Philadelphian, JB Lippincott, 1988, pp 1-612. which says more than 50% disease of orbit presents eccentrically,

supporting to make clinical diagnosis more easier by the nature of displacement and requesting further neuro imaging studies oriented to the suspected area of lesion.

Painful proptosis occurs mostly in orbital inflammatory lesion, primary tumors, metastasic lesion of orbit, accounting for 53% of our study which correlates with Jakobiec FA, Jones IS: Orbital inflammations. In Duane TD (ed): Clinical Ophthalmology, 2nd ed, New York, Harper & Row 1980, pp 1-75. . Rootman J: Diseases of the orbit . Philadelphian, JB Lippincott, 1988, pp 1-612. which shows 38% of cases presenting with pain.

Proptosis presents most commonly as gradual onset which is 49.8% in our study comparable with Shields JA, Bakewall B, Augsburger JI, et al: Classification and incidence of space –occupying lesions of the orbit: A survey of 645 biopsies. Arch Ophthalmol 102: 1606 – 1611, 1984. which shows 50% of cases of gradual onset of proptosis.

49.8% of our case present with exophthalmometry value of 3- 5mm, 29.66% presented with 6-10mm, 8.3% with 2mm, 3.3% with more than 10mm. Because various instruments are used in measurement of protopsis in cases of extra axial, subjective variation in measurement, placement of instrument in orbital margin varies, no study could be exactly compared. But Newton TH, Bilaniuk LT, eds Radiology of the eye & orbit (Modern neuro radiology) volume 4 New York Ravan; 1990 describes standard instruments pick out

lesion of 3-5mm more earlier, lesser than this lesions are too subtle , greater than this makes proptosis obvious.

Although globe is displaced binocularity is maintained between the two eyes in proptosis. Diplopia becomes a complaint only when cranial nerves are involved or when there is muscle restriction of particular movement in these patients and patients do not have very good clarity of vision due to optic nerve involvement , choroidal folds , macular edema. Diplopia was a complaint only in 10% of our cases.

Of the 60 cases 44 (73%) presented with primary orbital pathology and 16 (27%) presented with secondary orbital lesion consistent with Jakobiec FA, Jones IS: Orbital inflammations. In Duane TD (ed): Clinical Ophthalmology, 2nd ed, New York, Harper & Row 1980, pp 1-75. of 95 consecutive cases 87% were primary 13% were secondary.

18 (30%) of our cases were confirmed by histopathological examination 2(3.3%) of our cases were confirmed by doppler study remaining 40(66.4%) cases diagnosis conformed by clinical examination, radio imaging , therapeutic trial and observation. On reviewing the literature five major reviews two were based on pathology specimens (Wilsons and Grossniklaus and shields), one was based on a tumor registry (Kennedy) two were based on clinical orbital practice (Henderson and Rootman).

60 cases presented with unilateral proptosis out of 108 cases which is 55.5% which is in accordance with Jones IS, Jakobiec FA: diseases of the orbit. Hagerstown , MD, Harper & Row, 1979, pp 17-30. which shows 53% of unilateral protopsis

According to etiology our study is comparable with

Aetiology	No. of cases	Percentage	Jakobiec	Shields et al
Congenital	2	3.32	6%	9%
Inflammatory	23	38.18	45%	17%
Grave's disease	12	25	22%	38%
Vascular	2	3.32	3.3%	6%
Tumour	7	11.62	12%	11.5%
Sinuslesion	4	6.64	3%	3.2%
Metastasis	4	6.64	3%	4.5%
Orbital cellulites	3	4.98	3%	4.5%
Miscellaneous	3	4.98	3%	6.3%

Medical treatment was given for 22(36.52%) of patients, Medical and surgical procedures like incision and drainage, biopsy, FNAC was done for 11 (18.2%) cases, orbitotomy was done for 7 (11.62%) cases one of which underwent Radiotherapy, chemotherapy was given for 2patients, enucleation

was done for 2 cases, exenteration was done for 2 cases, Endonasal sinus surgery was done for 2 cases, 10(19.92%) were put under observation.

Of the 60 cases, 18 (29.88%) cases had complete resolution 12 (19.92%) were partly resolved, irregular treatment was taken up by 9 cases (14.94). 5 lost follow up. 2 patients underwent enucleation. 2 patients under exenteration. 2 patient died during our study 10patients were put underwent observation. Resolution was considered when patients were symptom free and exophthalmometry value comes with in normal limits.

Partial resolution was considered if symptoms were not relieved or if exophthalmometry value remained abnormal. Irregular treatment when the patient did not keep up to the regular dosage schedule as given by us. Palmer BW; Unilateral exophthalmos. Arch Ophthalmol 82: 415 1965. who showed total resolution of 30% of his cases, irregular treatment 16% , partially resolved 25% , lost followup 10% , death 5% of his cases.

At the end of two year period of our study 49.9% of our patients had recovered good vision which was 24.9% in the beginning of our study.

SUMMARY

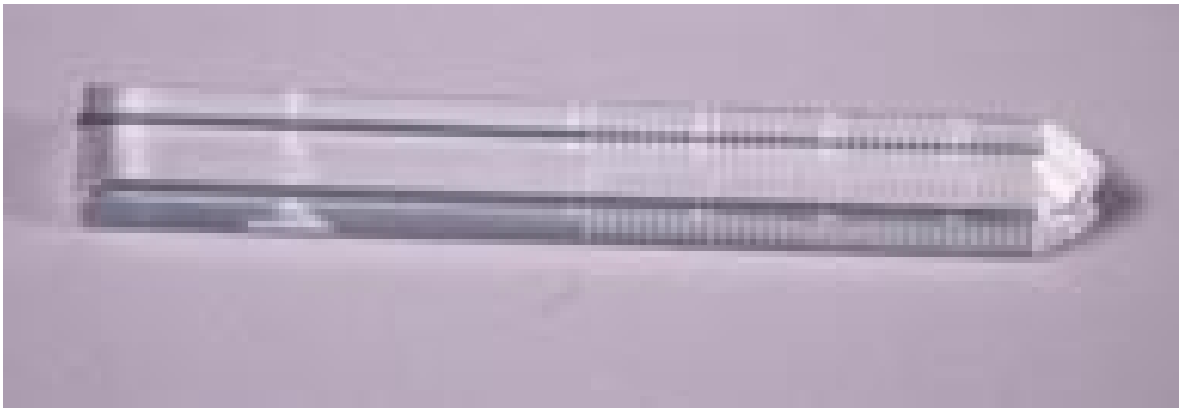
The literature on unilateral proptosis is reviewed. Materials and methods employed are stated. Sixty cases were studied out of whom 29 were males and 31 were females. Unilateral proptosis was the presenting feature in 5.55% of all the orbit clinic patients. Unilateral proptosis was five times more common than bilateral proptosis. Defective vision was the commonest associated symptom, which was present in 82% of the patients with unilateral proptosis. Pain was associated with 41.8 % of cases and Diplopia was present is only 10% of cases. Right eye was more commonly involved than the left eye. Eccentric proptosis was more common than axial proptosis. Irreversible optic nerve damage resulted in 11.62% patients. Inflammation was the commonest aetiology found in adults 38.18%. Thyroid opthalmopathy comes next with 25%. Neoplasm was 11.62%. 100% cure rate was achieved in vascular lesions. Prognosis was second best in inflammatory lesions. The cure rate being 85%. Children responded better than adults to treatment. Complete visual recovery was attained with vascular lesions after surgery. Visual recovery in inflammatory lesions was 73%. Primary orbital pathology responded well to treatment than secondary involvement. Biopsy was found to be an useful adjunct to x- rays and CT scan.

CONCLUSION

In Conclusion orbital disease offers clinicians a challenging field in which to pursue diagnostic, medical, and surgical modalities. Inflammatory orbital disease accounts for leading cause of orbital disorders followed by thyroid ophthalmopathy. Neurogenic, and vascular mass lesions are the common tumor subdivisions in both adults and children. Fortunately, orbital mass lesions are benign in majority of cases, but life-threatening malignancy must always be considered.

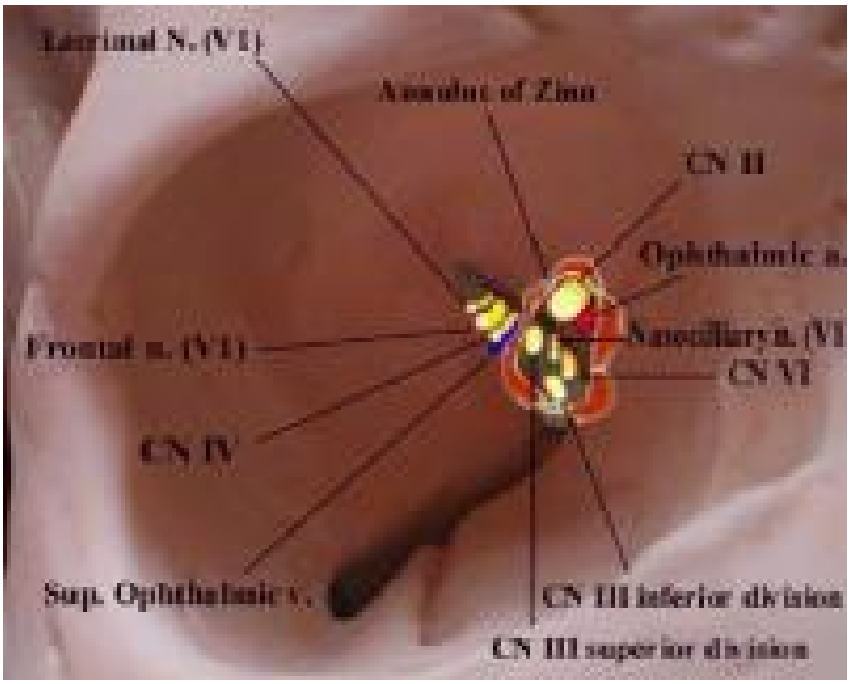
EXOPHTHALMOMETERS

LUEDDELS

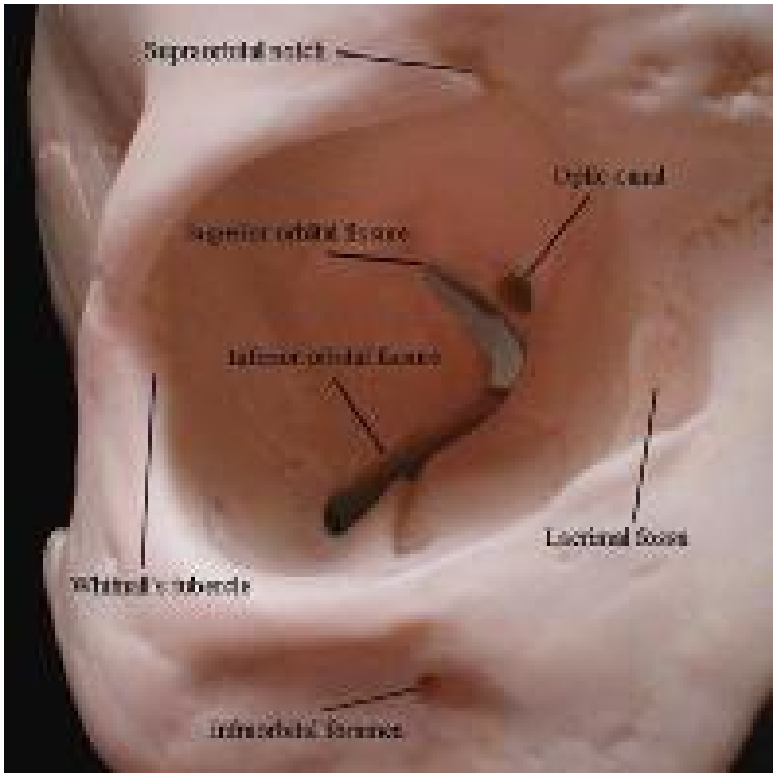


HERTELS

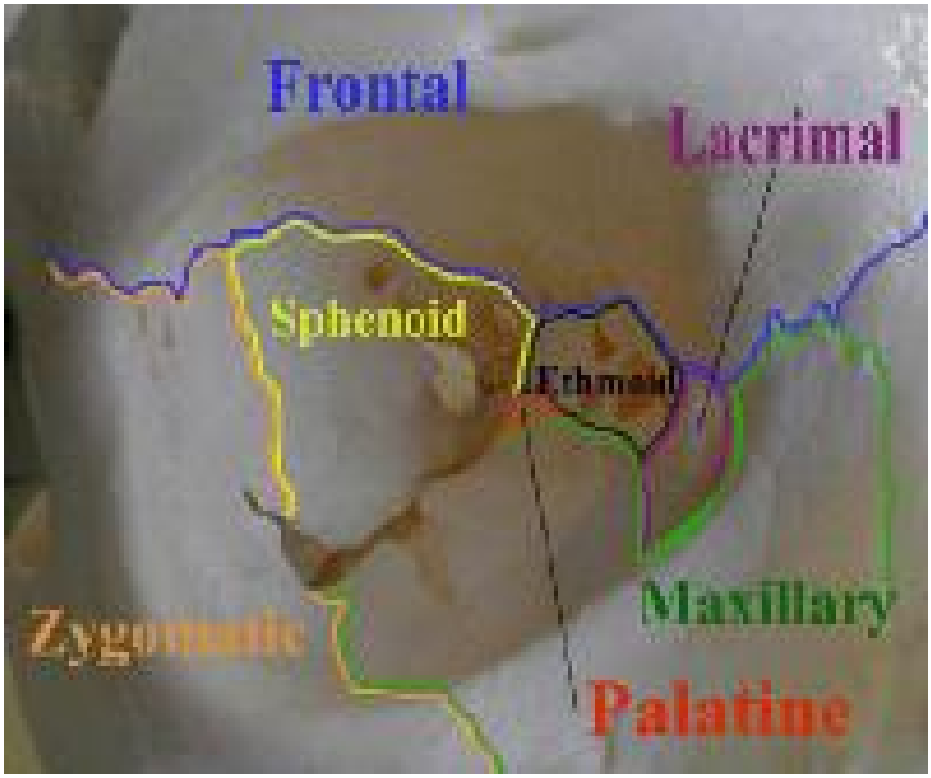
ORBITALAPEX



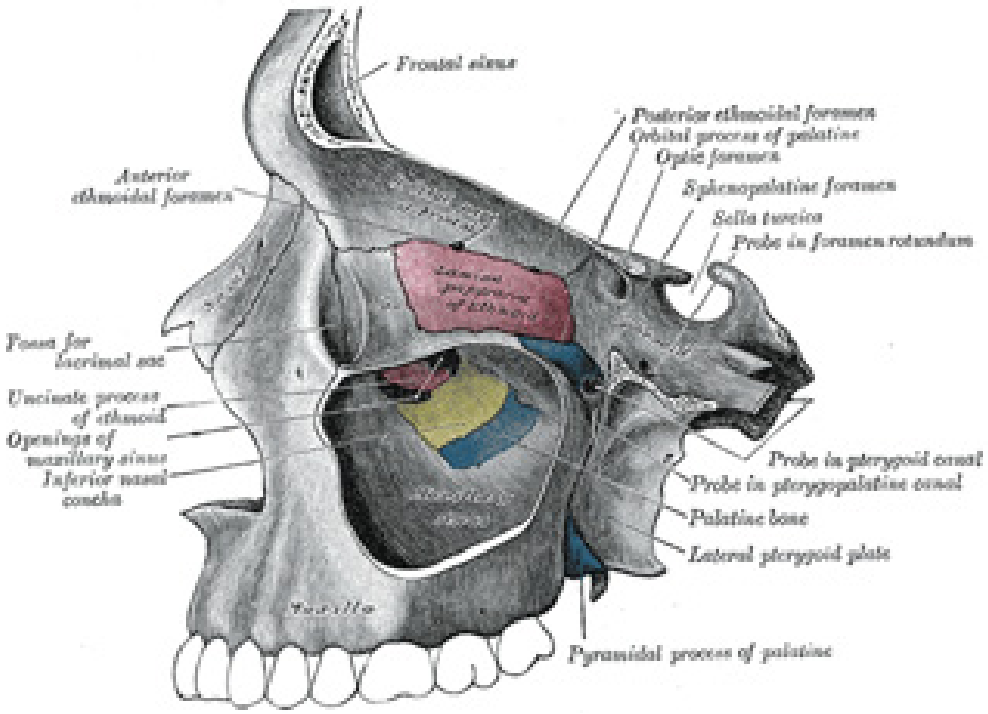
ORBITAL FISSURES



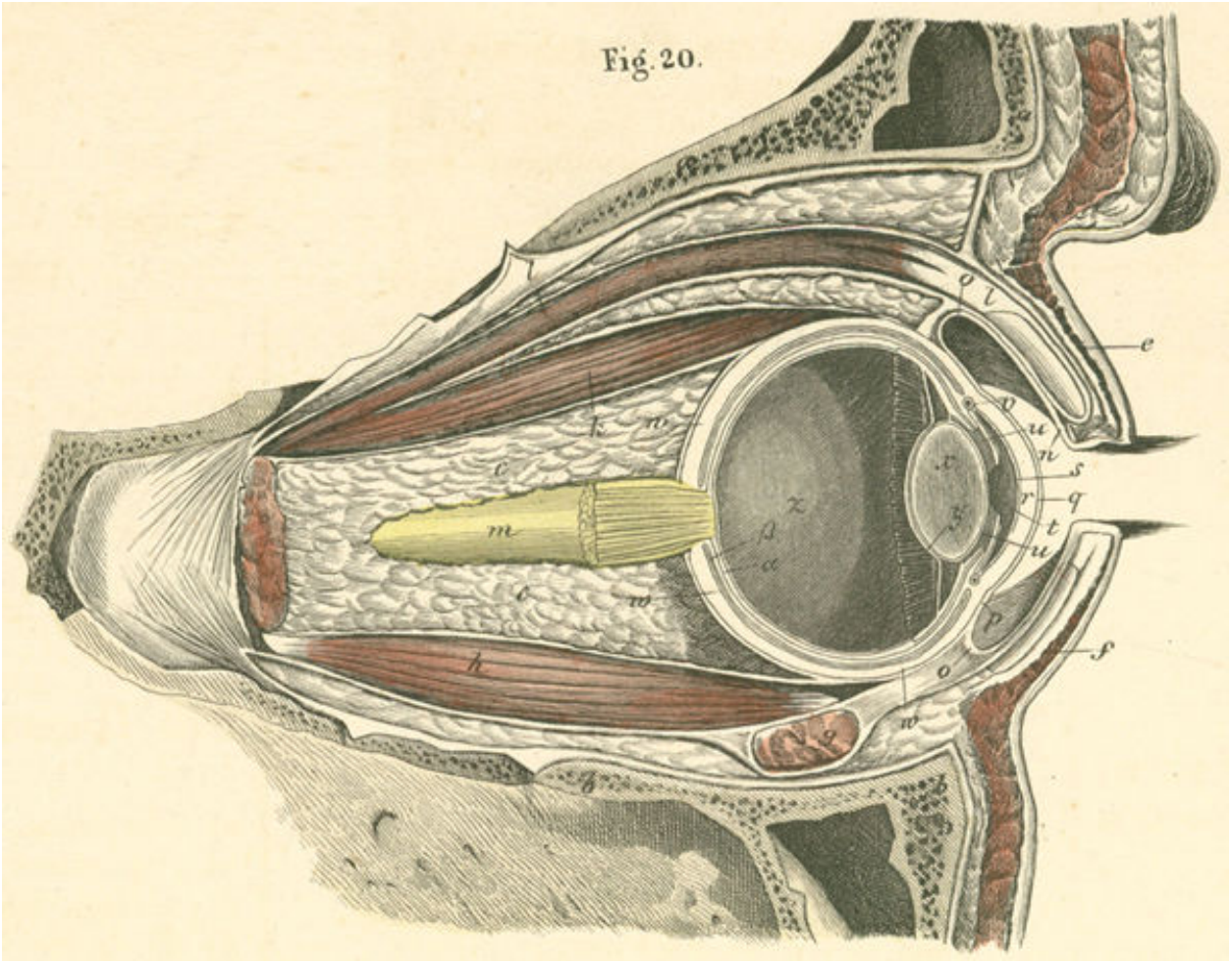
WALLS OF ORBIT



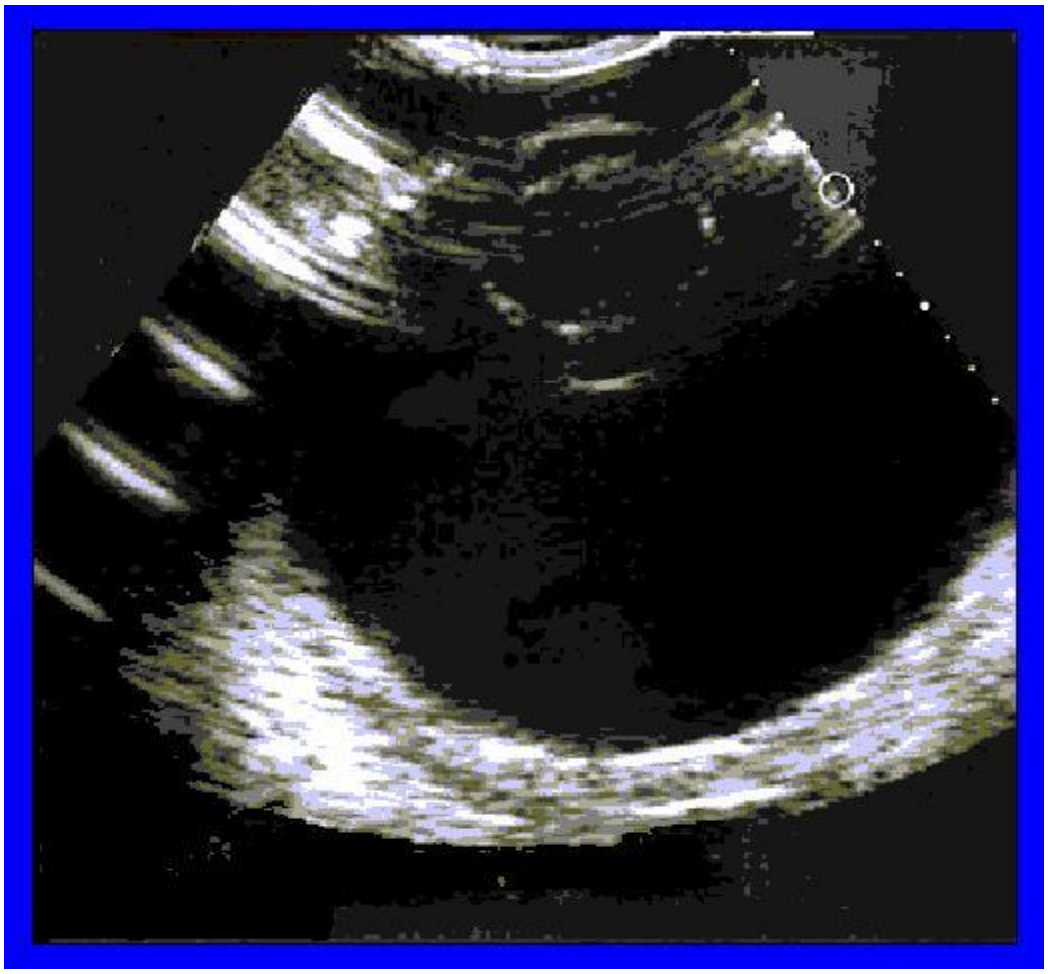
RELATION TO SINUSES



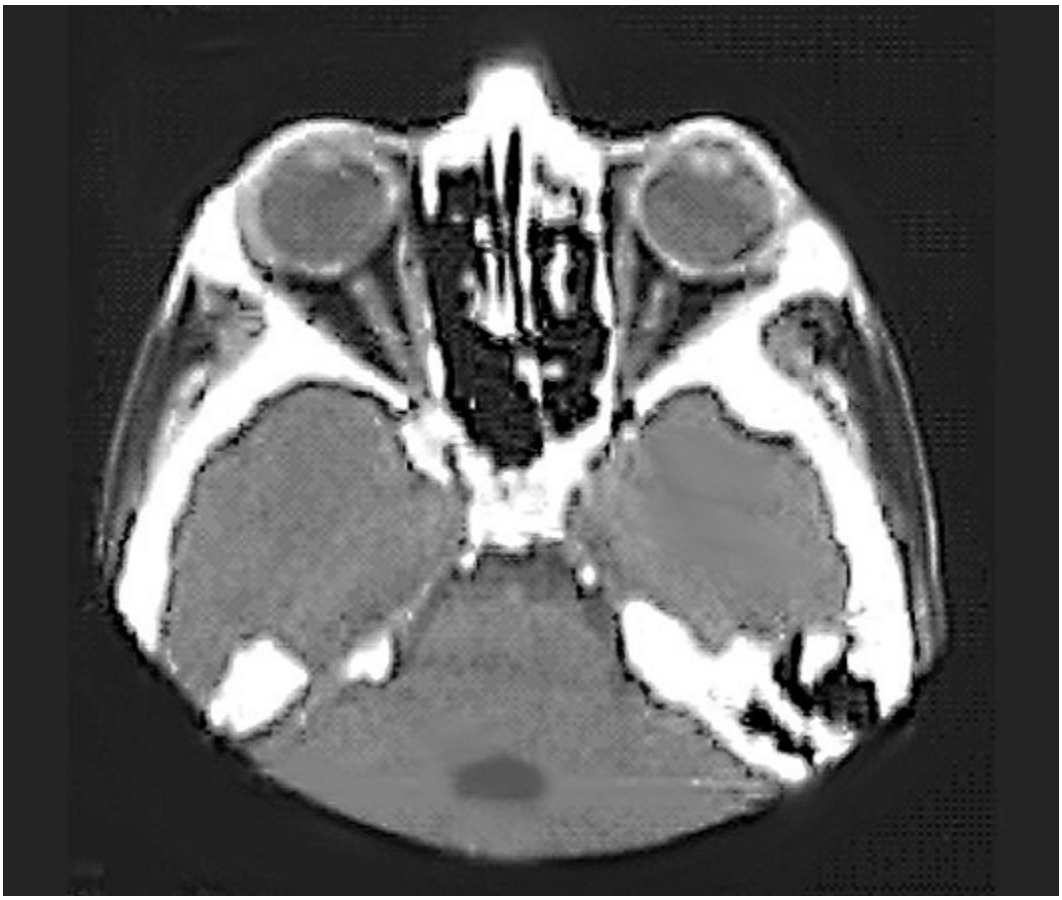
ORBITAL SPACES



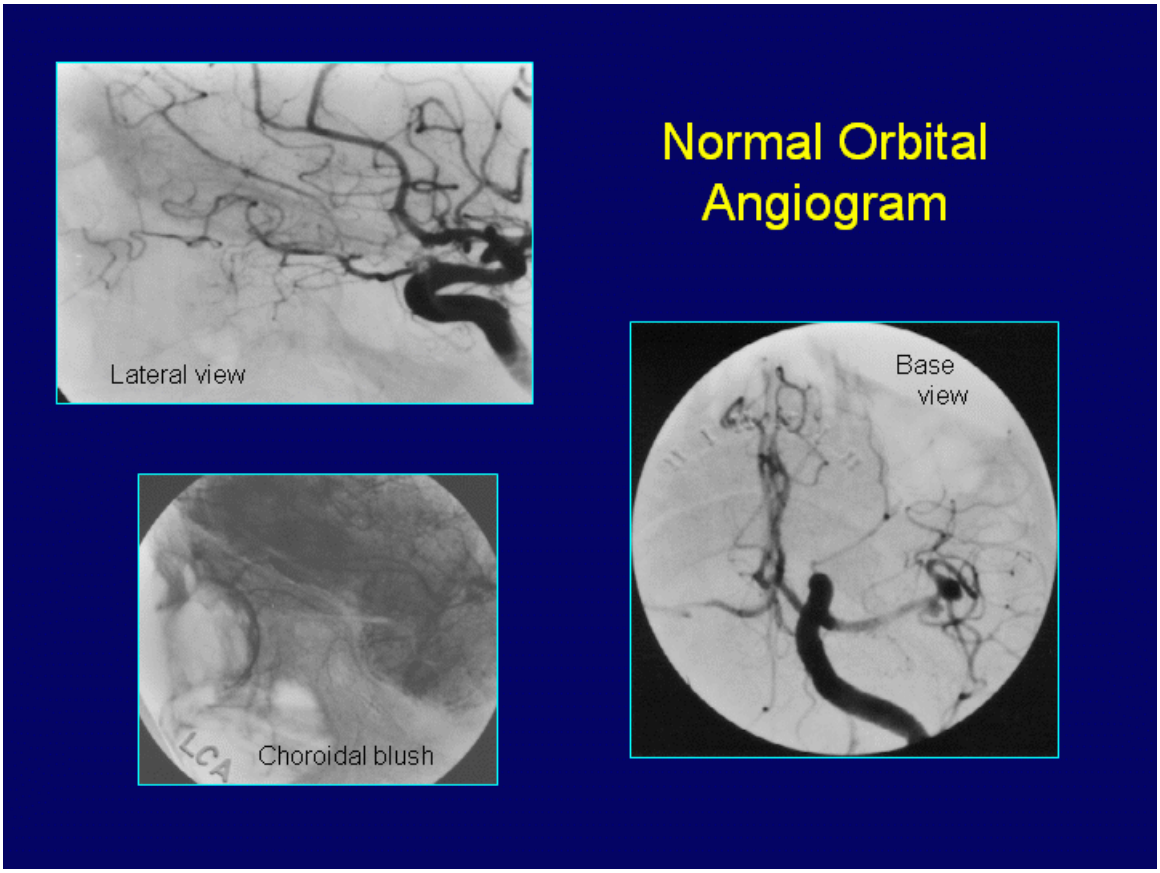
B- SCAN



CT SCAN – AXIAL VIEW

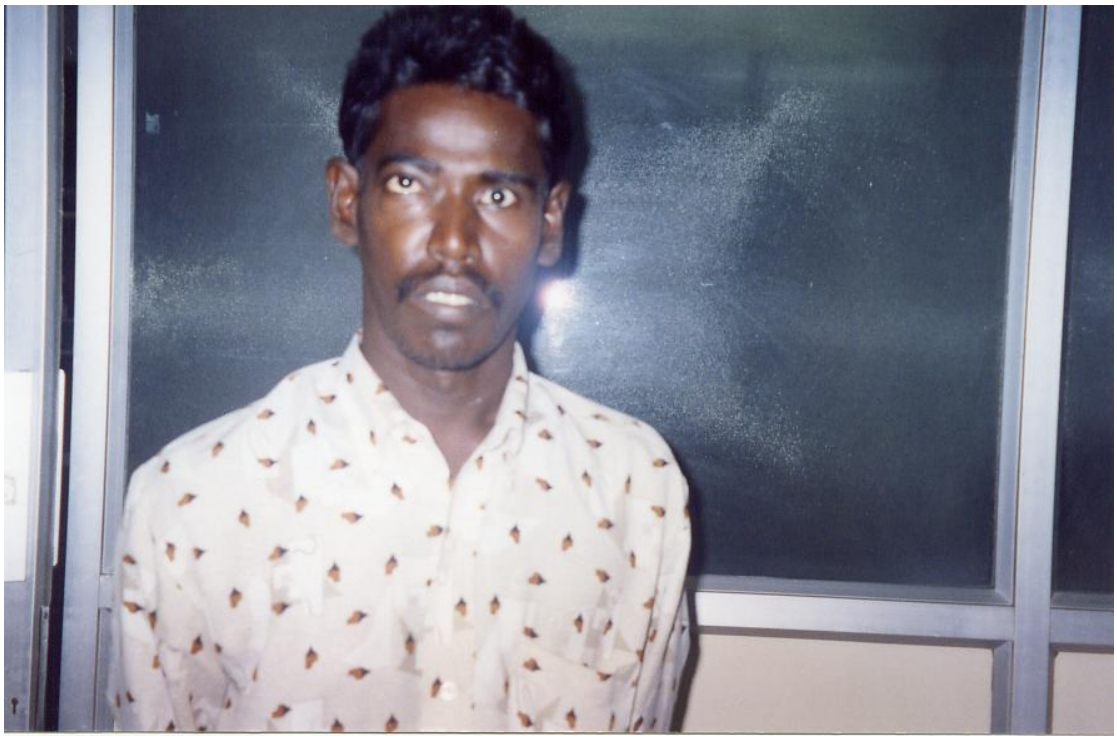


ORBITAL ANGIOGRAM



LACRIMAL GLAND TUMOUR

MAXILLARY SINUS CARCINOMA



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PROFORMA

Name : Age : SL.No .

Address : Sex :

O.P.No / I.P.No: Occupation :

COMPLAINTS **DURATION**

1. Proptosis :
2. Defective vision :
3. Pain :
4. Diplopia :

Present History

Onset since birth / sudden / gradual

Nature of Progression

Past history

Personal history Non-Veg / Veg

General Examination

Build:

B.P.

Pulse :

Lymphnodes :

Thyroid :

Ocular examination Right left

Lids :

Lacrimal gland:

Lacrimal sac :

E.O.M :

Conjunctiva :

Cornea : sensation

Ulcer

Anterior Chamber :

Iris :

Pupil :

Lens :

Vision :

Retinoscopy :

Tension :

N.L. Duct :

Fields :

Fundus :

EXOPHTHALMOMETRY

PROPTOSIS:

Axial / Eccentric

Visible mass : Yes / No

Tenderness : Yes / No

Reducibility Yes / No

Pulsatile Yes / No

Cough impulse : Yes / No

Valsalva: Increase / Negative

Thrill: Yes / No

Bruit: yes / No

SYSTEMIC

CVS:

RS:

CNS:

ENT:

SKIN:

OTHERS:

INVESTIGATIONS

Blood: Routine

Immature cells:

Urine:

VDRL:

Mantaoux:

T_3, T_4:

X-ray:

Orbit, Sinuses, Chest, Skull

A – scan:

B – scan:

C T – scan:

Doppler:

TREATMENT

MEDICAL

SURGICAL

RADIATION

CHEMOTHERAPY

BIOPSY

PROGRESS

ABBREVATIONS

CCF – Carotid Cavernous Fistula

CNS – Central Nervous System

CVS – Cardio Vascular System

ESR – Erythrocyte Sedimentation Rate

ETH – Ethmoidal Sinus

FNAC – Fine Needle Aspiration Cytology

LG – Lacrimal Gland

OM – Occipito Mental

OF – Occipito Frontal

PNS – Para Nasal Sinus

SOL – Space Occupying Lesion

SOV – Superior Ophthalmic Vein

SPH – Sphenoidal Sinus

TSH – Thyroid Stimulating Harmone

CODE FOR MASTER CHART

ANTERIOR SEGMENT

Lids	Normal	LCN
	Edema (+)	LCE
Cornea	Normal	CN
	Exposure keratitis	CE
Pupil	Normal	PN
	RAPD	PR

OCULAR MOVEMENTS

Full	F
Restricted	R

FUNDUS

N	Normal
DV	Dilated Vessel
SOA	Optic Atrophy

TREATMENT

M	Medical
M + S	Medical (+) Surgical
S	Surgical
SN	Enucleation
SE	Exenteration
OR	Orbitotomy
OR + RT	Orbitotomy (+) Radiotherapy
CHEMO	Chemotherapy

DIAGNOSIS

C	Congenital
I	Inflammation
TH	Thyroid
V	Vascular
T	Tumor

T_{RAB}	Rhabdomyosarcoma
T_{AMF}	Angiomyofibroma
T_{LY}	Lymphoma
T_{LA}	Lacrimal gland tumor
T_{D}	Dermoid
S	Sinus
M	Metastasis
OC	Orbital cellulites
MIS	Miscellaneous

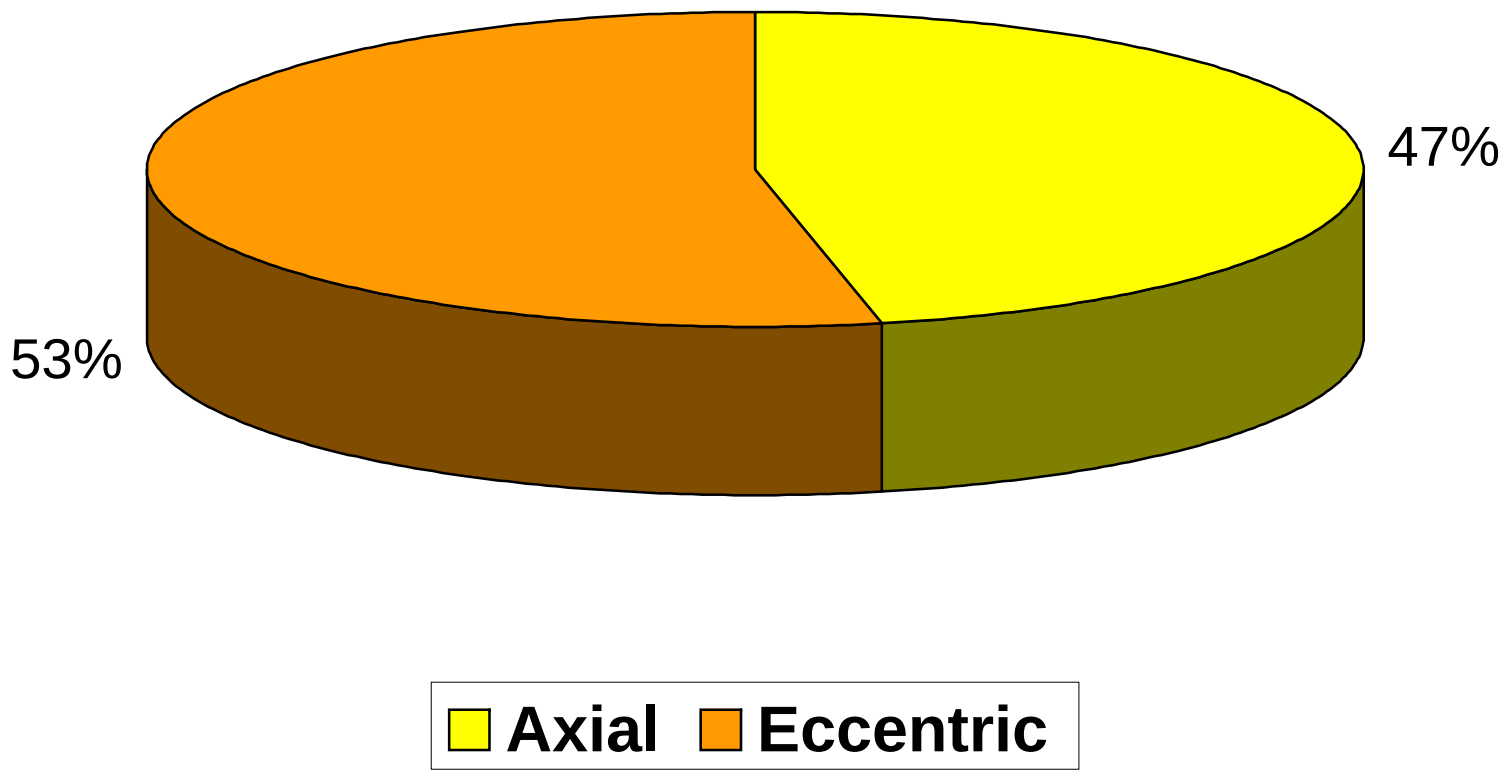
TREATMENT

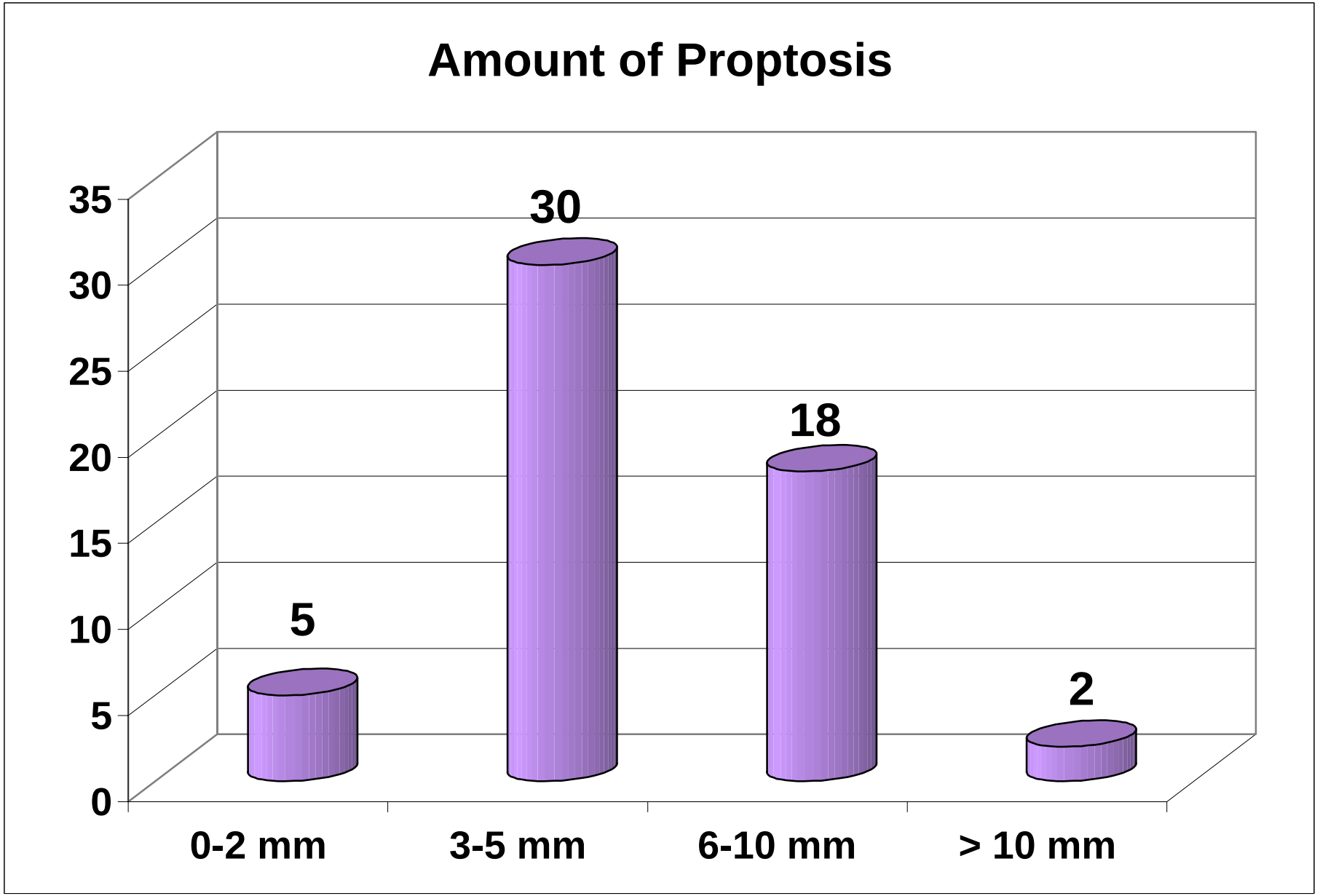
O	Observation
M	Medical
M+S	Medical + Surgical
OR	Orbitotomy
Chemo	Chemotherapy
EN	Enucleation
EX	Exenteration
ENS	Endonasal surgery

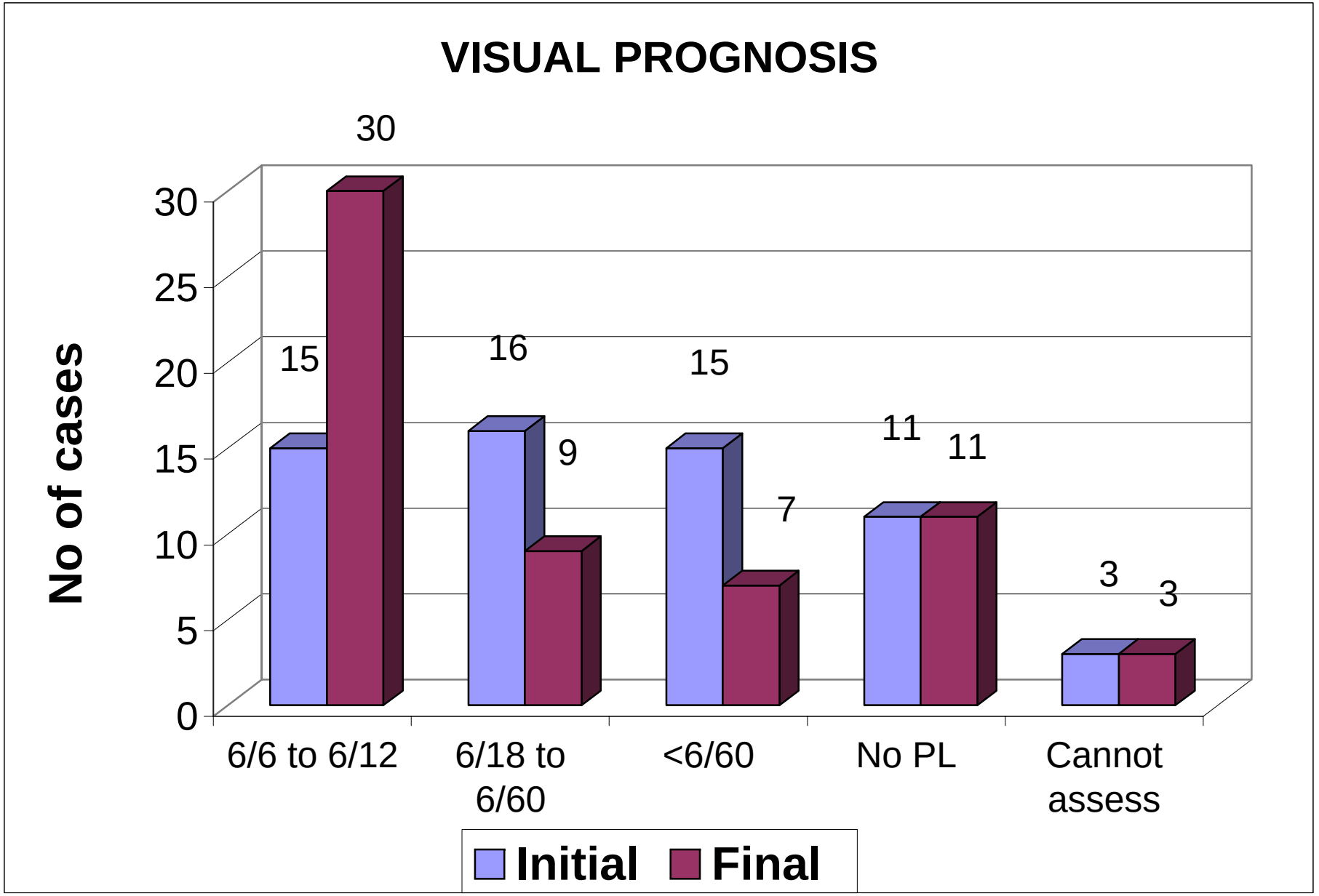
PROGRESS

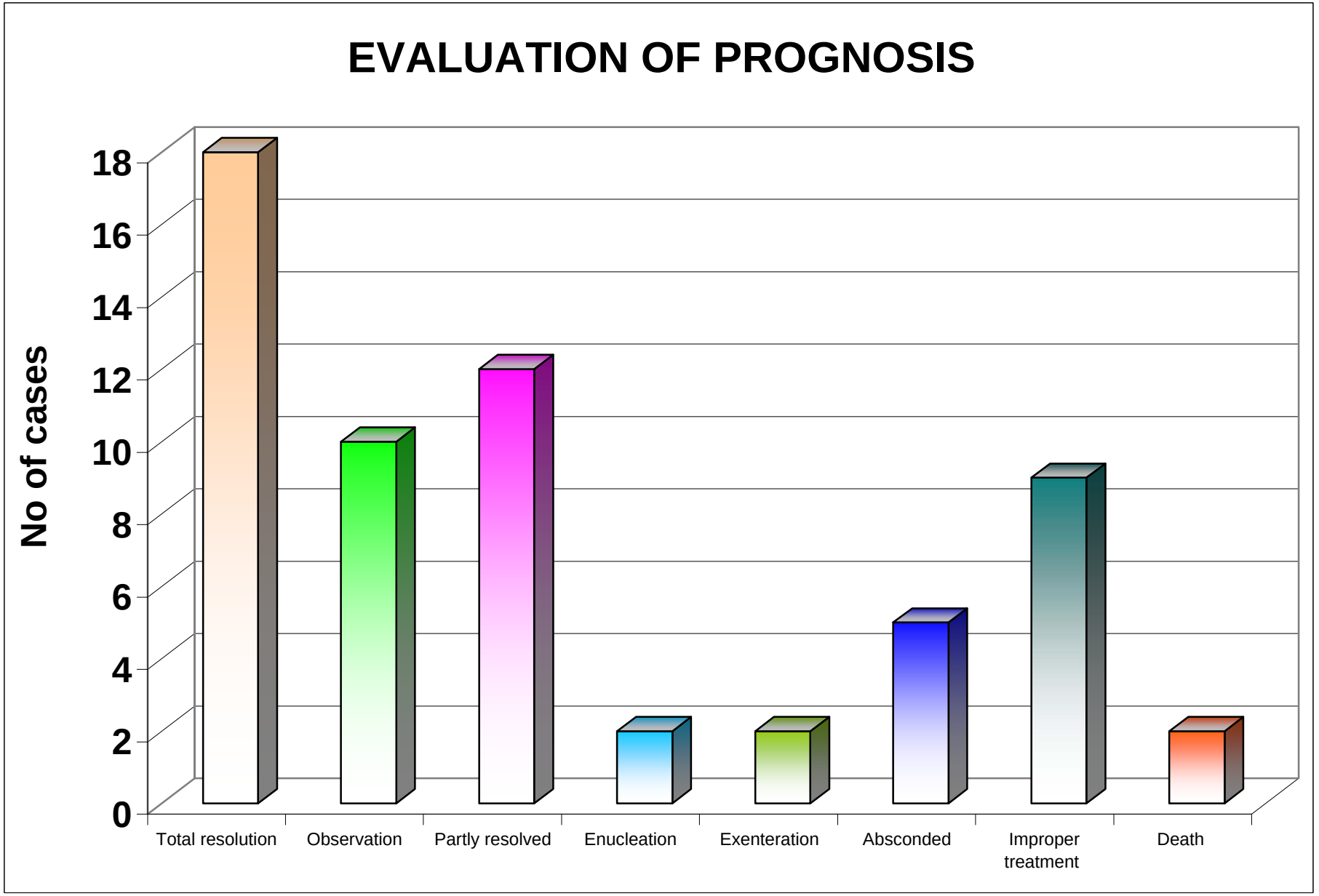
R	Recovery
O	Observation
L	Lost follow up
EN	Enucleation
EX	Exenteration
PR	Partly Resolved
UR	Un Resolved – poor compliance
D	Death

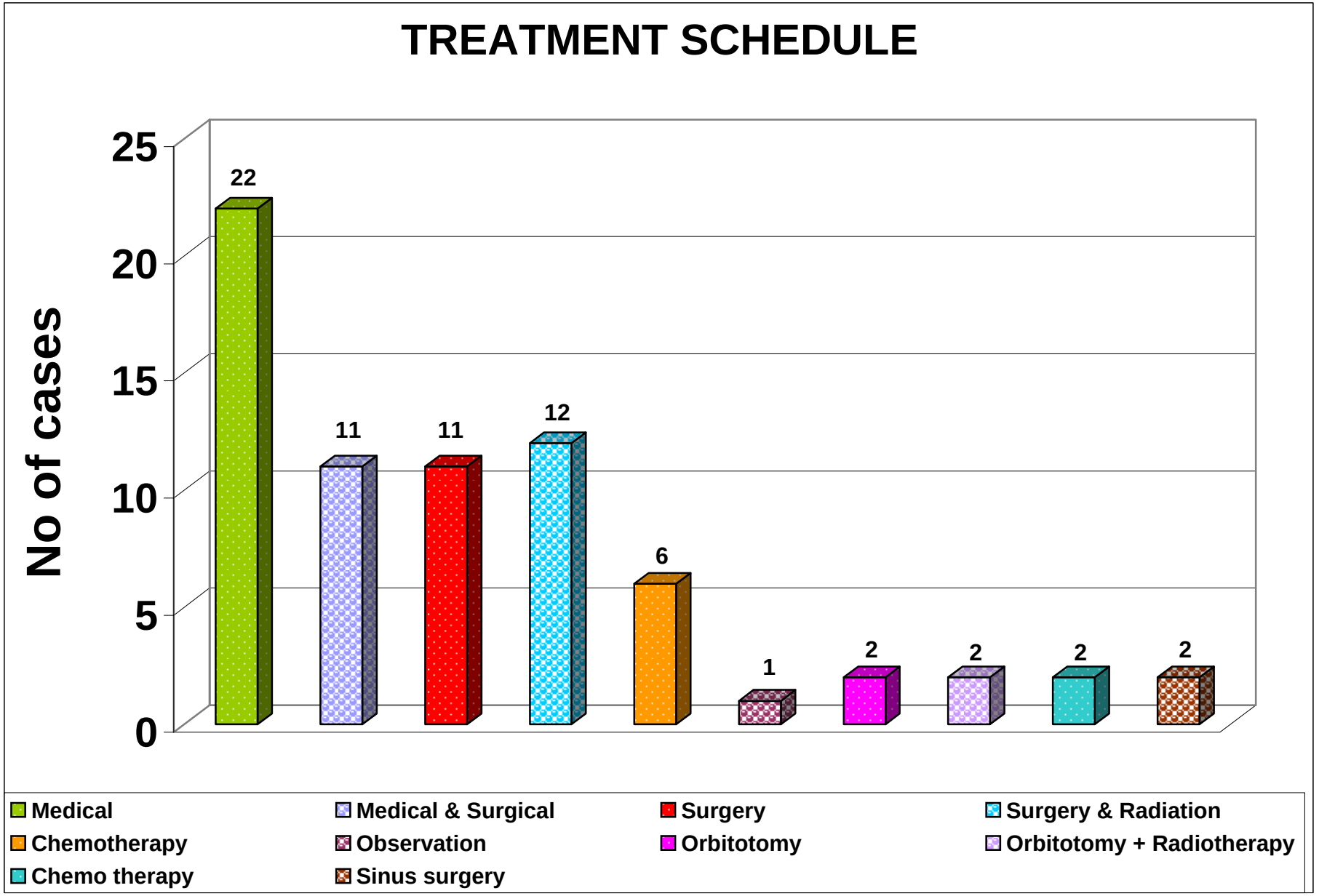
THE TYPE OF PROPTOSIS ENCOUNTERED




Amount of Proptosis
35
30
25
20
15
10
5
0
30
18
5
2
0-2 mm
3-5 mm
6-10 mm
> 10 mm


VISUAL PROGNOSIS
No of cases
30
25
20
15
10
5
0
15
30
16
9
15
7
11
11
3
3
6/6 to 6/12
6/18 to 6/60
<6/60
No PL
Cannot assess
Initial
Final


EVALUATION OF PROGNOSIS
No of cases
18
16
14
12
10
8
6
4
2
0
Total resolution
Observation
Partly resolved
Enucleation
Exenteration
Absconded
Improper treatment
Death


TREATMENT SCHEDULE
No of cases
25
20
15
10
5
0
22
11
11
12
6
1
2
2
2
2
Medical
Medical & Surgical
Surgery
Surgery & Radiation
Chemotherapy
Observation
Orbitotomy
Orbitotomy + Radiotherapy
Chemo therapy
Sinus surgery

MASTER CHART

S.No	Name	Age	Sex	Side Involved	Proptosis	Ant . Seg	OM RL	Vision intially	Final	Fundus of involved eye	Exophthal mometry in mm		Diagnosis	Treatment	Recovery	Other systems
											R	L				
1	Raja	81	M	RE	EA	LCE	R	<6/60	6/24	N	22	18	OC	M	R	
2	Ramani	72	F	LE	A	CE	R	No PL	No PL	OA	15	19	M	O	O	
3	Ravi	3	M	RE	A	LCE CE	F	C.A	C.A	N	NP	NP	C	O	O	Breast ca
4	Pillai	82	F	LE	EA	LN CN	R	No PL	No PL	OA	15	19	M	Chemo	L	
5	Geetha	5	M	RE	A	LCE CE	F	C.A	C.A	N	NP	NP	C	O	O	
6	Ponnu	75	F	LE	EA	LNCN	R	6/36	6/24	N	20	23	OC	M+S	R	
7	Elanchi	7	M	RE	A	LCE CE	R	No PL	No PL	OA	21	16	TG	EN	EN	
8	Gomathi	76	F	LE	EA	CE	F	6/36	6/36	N	17	20	I	M+S	PR	
9	Gowtham	9	M	RE	EA	LCE CE	F	No PL	No PL	OA	20	15	M	Chemo	D	Adrenal NB
10	Ganaga	73	F	LE	EA	LCE CE	F	<6/60	C.A	N	18	21	OC	M+S	PR	
11	Mani	11	M	RE	A	CE PR	R	6/12	6/12	N	NP	NP	TG	EN	EN	
12	Nesi	6	F	RE	EA	CE PR	F	No PL	6/6	OA	21	16	TRAB	OR+RT	L	
13	Kutti	13	M	RE	EA	LC CE	F	C.A	6/9	N	NP	NP	M	O	O	
14	Kanga	63	F	RE	A	LCN CN PN	F	<6/60	6/36	N	22	18	I	M+S	R	
15	Kannan	15	M	LE	EA	LCN CN PN	F	6/12	6/9	N	NP	NP	TD	OR	UR	
16	Kaveri	62	F	LE	A	LCN CN PN	F	<6/60	6/12	N	19	22	I	M+S	R	
17	Murugan	17	M	RE	A	LCN CN PN	F	6/36	6/12	N	19	15	I	O	UR	
18	Subramani	64	M	LE	EA	LCN CN PN	F	<6/60	No PL	N	18	21	I	M+S	R	
19	Pooranan	19	M	RE	A	LCN CN PN	R	6/36	6/12	N	22	16	S	ENS	PR	ENT
20	Chinnathatee	66	F	LE	EA	LCN CN PN	F	6/24	6/12	N	18	21	TH	M	UR	TH
21	Rameesha	21	F	RE	EA	CE PR	F	No PL	No PL	OA	21	16	TAMF	OR	L	
22	Palani	68	M	LE	A	LCN CN PN	F	6/18	6/12	N	16	19	I	M	UR	
23	Nagasamy	23	M	RE	EA	LCN CN PN	F	<6/60	6/36	N	23	18	S	ENS	PR	ENT
24	petchiamma	67	F	LE	EA	LCN CN PN	F	6/36	6/24	N	20	23	TH	M	PR	TH
25	Nagarajan	25	M	RE	EA	CE PR	R	6/18	6/19	N	20	17	I	M	UR	
26	Panchaloon	63	F	LE	A	LCN CN PN	F	No PL	No PL	OA	18	21	TLY	OR	D	
27	Gurusamy	27	M	RE	EA	LCN CN PN	R	6/12	6/12	DV	20	17	V	OR	R	
28	Pandiyamma	68	F	LE	A	LCN CN PN	F	6/9	6/9	N	17	20	TH	M	L	TH

29	Guru	29	M	RE	EA	CE	F	<6/60	6/9	N	21	16	MIS	M+S	PR	
30	Menaka	30	F	LE	EA	LCN CN PN	F	6/12	6/9	N	18	21	I	M	PR	
31	Solai mani	34	F	RE	EA	LCN CN PN	F	<6/60	6/12	N	19	15	TH	M	R	TH
32	Moorthy	40	M	LE	A	LCN CN PN	F	6/36	6/9	N	18	21	I	M	R	
33	Santha	50	F	RE	EA	LCN CN PN	F	6/24	6/9	N	22	16	TH	M	R	TH
34	Ramesh	35	M	LE	A	LCN CN PN	F	6/18	6/9	DV	20	23	V	OR	R	
35	Rathna	36	F	RE	A	LCN CN PN	F	6/9	6/9	N	20	16	I	M	PR	
36	Kumaran	39	M	LE	EA	LCE CE PR	F	<6/60	No PL	N	17	20	S	EX	EX	ENT
37	Pansitham	31	F	RE	EA	LCN CN PN	F	6/12	6/9	N	18	15	I	M	R	
38	Murugan	33	M	LE	A	LCN CN PN	F	6/9	6/6	N	15	18	I	M	R	
39	Pyori john	38	M	RE	EA	LCN CN PN	F	6/36	6/12	N	20	17	I	M	R	
40	Senthil	37	F	LE	A	LCN CN PN	F	6/24	6/24	N	16	19	I	M+S	UR	
41	Sikkandar	49	M	RE	A	LCN CN PN	F	<6/60	6/12	N	21	16	MIS	M	R	
42	Senthil	46	F	LE	EA	LCN CN PN	F	6/12	6/12	N	17	20	I	M+S	UR	
43	Viji	40	M	RE	A	LCN CN PN	F	6/24	6/9	N	22	19	I	M	L	
44	Angusamy	45	F	LE	EA	LCN CN PN	F	6/9	6/6	N	18	21	I	M	R	
45	Annad	43	M	RE	A	CE	F	<6/60	6/12	N	23	18	I	M	R	
46	Dinesh	41	F	LE	EA	LCN CN PN	F	6/36	6/12	N	19	22	I	O	R	
47	Vetiyan	47	M	RE	A	LCN CN PN	F	6/12	6/6	N	21	18	I	O	O	
48	guruvuamma	42	F	LE	A	CE	F	<6/60	6/9	N	20	23	TH	M	O	TH
49	Anand	43	M	RE	EA	CE	F	<6/60	6/36	N	23	20	I	O	UR	
50	Pancha varanam	50	F	LE	A	LCN CN PN	F	6/9	6/9	N	15	18	TH	O	O	TH
51	Bala	59	M	RE	EA	LCN CN PN	F	6/24	6/12	N	22	19	I	M	R	
52	Jayo	57	F	LE	A	LCN CN PN	F	6/12	6/9	N	16	19	TH	O	O	TH
53	Balaguru	55	M	RE	EA	LCN CN PN	F	6/36	6/18	N	20	17	I	M	PR	
54	Jothi	53	F	LE	A	LCN CN PN	F	6/9	6/9	N	17	20	TH	O	O	TH
55	Thangaraj	51	M	RE	EA	LCN CN PN	F	6/12	6/12	N	19	16	TH	O	O	TH
56	Petchi	54	F	LE	EA	CE PR	R	No PL	No PL	N	15	24	TLG	OR	PR	
57	Muniamma	52	F	RE	A	LCN CN PN	F	6/9	6/9	OA	18	21	TH	O	PR	
58	Andichi	56	F	LE	EA	LCE CE PR	R	No PL	No PL	N	16	25	S	EX	EX	ENT
59	Mayil	60	F	RE	A	LCN CN PN	F	<6/60	6/36	OA	19	22	MIS	M+S	PR	
60	Santhiri	58	F	LE	A	CE	F	<6/60	6/36	N	20	23	TH	M	UR	TH